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Health Effects Criteria for Marine Recreational Waters

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Research Triangle Park, NC

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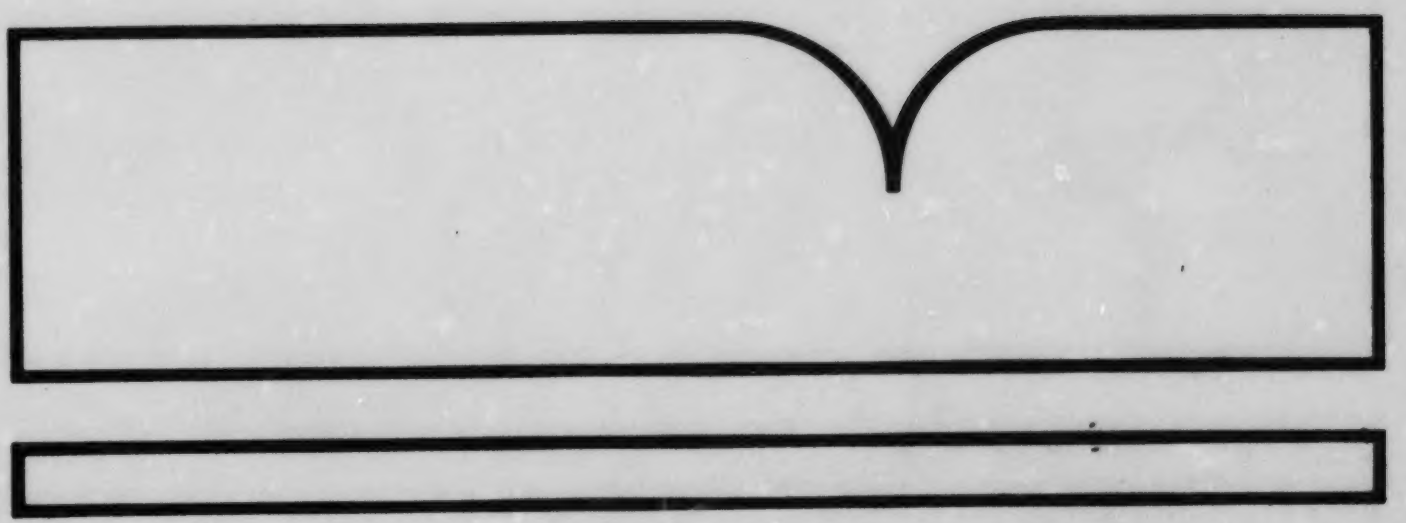

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16. ABSTRACT This report presents health effects quality criteria for marine recreational waters and a recommendation for a specific criterion among those developed. It is the mathematical relationship of the swimming-associated rate of gastrointestinal symptoms among bathers to the quality of the water as determined by the density of a fecal indicator, enterococci. Thus, it can be used to provide guidelines based upon acceptable rather than detectable risk and is consistent with risk analysis.

The criteria were developed using data collected from an extensive in-house extramural, microbiological-epidemiological research program conducted by the U.S. Environmental Protection Agency over the years 1972-1979. Central to this program was the conduct of prospective epidemiological-microbiological studies using a design developed at the Marine Field Station of the Health Effects Research Laboratory. These multi-year studies were conducted at beaches at 3 locations in the U.S. An additional study was conducted in Alexandria, Egypt; however, for the reasons given, only the U.S. data were used in the development of the criteria.

The two input parameters to the recommended model(criterion), the type of symptomatology and the specific water quality indicator, were determined from the analysis of data with a design which considered a number of symptom types and potential indicators. In addition, swimming was carefully defined as the exposure of the head to the water, the non-swimming controls were at the beach, and the trials were conducted over relatively short periods of time (1-2 days).

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HEALTH EFFECTS CRITERIA FOR MARINE RECREATIONAL WATERS

by

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FOREWORD

The many benefits of our modern, developing, industrial society are accompanied by certain hazards. Careful assessment of the relative risk of existing and new man-made environmental hazards is necessary for the establishment of sound regulatory policy. These regulations serve to enhance the quality of our environment in order to promote the pubic health and welfare and the productive capacity of our Nation's population.

The complexities of environmental problems originate in the deep interdependent relationships between the various physical and biological segments of man's natural and social world. Solutions to these environmental problems require an integrated program of research and development using input from a number of disciplines. The Health Effects Research Laboratory, Research Triangle Park, NC and Cincinnati, OH conducts a coordinated environmental health research program in toxicology, epidemiology and clinical studies using human volunteer subjects. Wide ranges of pollutants known or suspected to cause health problems are studied. The research focuses on air pollutants, water pollutants, toxic substances, hazardous wastes, pesticides and nonionizing radiation. The laboratory participates in the development and revision of air and water quality criteria and health assessment documents on pollutants for which regulatory actions are being considered. Direct support to the regulatory function of the Agency is provided in the form of expert testimony and preparation of affidavits as well as expert advice to the Administrator to assure the adequacy of environmental regulatory decisions involving the protection of the health and welfare of all U.S. inhabitants.

This report provides an assessment of the relationship between microbiological indicators of water quality and illness that may have resulted from swimming. The data base resulted from a series of in-house and extramural epidemiological-microbiological research projects designed to develop the criterion for marine waters. The development and periodic reevaluation of such criteria is mandated by Section 304(a)1 of Public Law 92-500: Federal Water Pollution Control Act Amendments of 1972; Clean Water Act of 1977.

F. Gordon Hueter, Ph.D.
Director
Health Effects Research Laboratory

PREFACE

Shortly after they were published by the National Technical Advisory Committee to the Federal Water Pollution Control Administration in 1968, the microbiological guidelines for direct contact recreational waters were attacked as being too restrictive. The basis for the attack was the meager and questionable epidemiological data from which they were derived, limitations of the microbial indicator of water quality (fecal coliforms) to be used, and defects in the methodology available for monitoring environmental waters for its presence. It was noted that these guidelines were recommended in the face of seemingly conflicting epidemiological findings from the studies conducted by Stevenson and Moore and a very limited number of outbreaks of infectious disease clearly shown to be associated with swimming in sewage polluted waters.

Early in 1969, it was suggested to the author of this report that he "look into the matter." During 1969 and early 1970, he and his colleagues developed a design for a prospective epidemiological-microbiological study differing from that used by Stevenson in a number of essential ways. A decision was made to look first at saltwater and later at freshwater beaches, and some beaches in New York City were identified for the conduct of a study.

The project was established in 1972 with a target date for completion in 1978-79. Studies were to be conducted at beaches in a number of locations in addition to New York City. The objective of the program was to produce criteria, defined as a mathematical relationship of some untoward effect from swimming in sewage polluted water to the quality of that water as measured by any of a number of potential microbial or chemical indicators; thus, they were to be amenable to risk analysis. The objective was achieved, and this report documents the output from that effort.

In addition, methods were developed and published for a rather large number of potential water quality indicators, and information and methodology were generated and published relative to several other problems in human infectious disease potentially or actually resultant from pollution of marine and fresh recreational waters. Included are the discharge of *Klebsiella* in industrial effluents, the relationship of *Aeromonas hydrophila, Acinetobacter* sp., *Pseudomonas aeruginosa,* and *Vibrio parahaemolyticus* densities to nutrient enrichment of aquatic environments, the potential for individuals to become colonized by multiantibiotic resistant coliforms via their activities in sewage polluted waters, the effect of environmental parameters on the survival of human pathogens and indicator microorganisms in marine and fresh waters, transfer frequencies for multiple antibiotic resistance into fecal isolates of *E. coli,* the characterization of a highly chlorine resistant, male specific coliphage from sewage, and the microbial colonization of the external ear canal.

ABSTRACT

This report presents health effects quality criteria for marine recreational waters and a recommendation for a specific criterion among those developed. It is the mathematical relationship of the swimming-associated rate of gastrointestinal symptoms among bathers to the quality of the water as determined by the density of a fecal indicator, enterococci. Thus, it can be used to provide guidelines based upon acceptable rather than detectable risk and is consistent with risk analysis.

The criteria were developed using data collected from an extensive in-house extramural, microbiological-epidemiological research program conducted by the U.S. Environmental Protection Agency over the years 1972-1979. Central to this program was the conduct of prospective epidemiological-microbiological studies using a design developed at the Marine Field Station of the Health Effects Research Laboratory. These multi-year studies were conducted at beaches at three locations in the United States (New York City, NY; Lake Pontchartrain, New Orleans, LA; and Boston Harbor, MA). An additional study was conducted in Alexandria, Egypt; however, for the reasons given, only the United States data were used in the development of the criteria.

The two input parameters to the recommended model (criterion), the type of symptomatology and the specific water quality indicator, were determined from the analysis of data with a design which considered a number of symptom types and potential indicators. In addition, swimming was carefully defined as the exposure of the head to the water, the non-swimming controls were at the beach, and the trials were conducted over relatively short periods of time (1-2 days).

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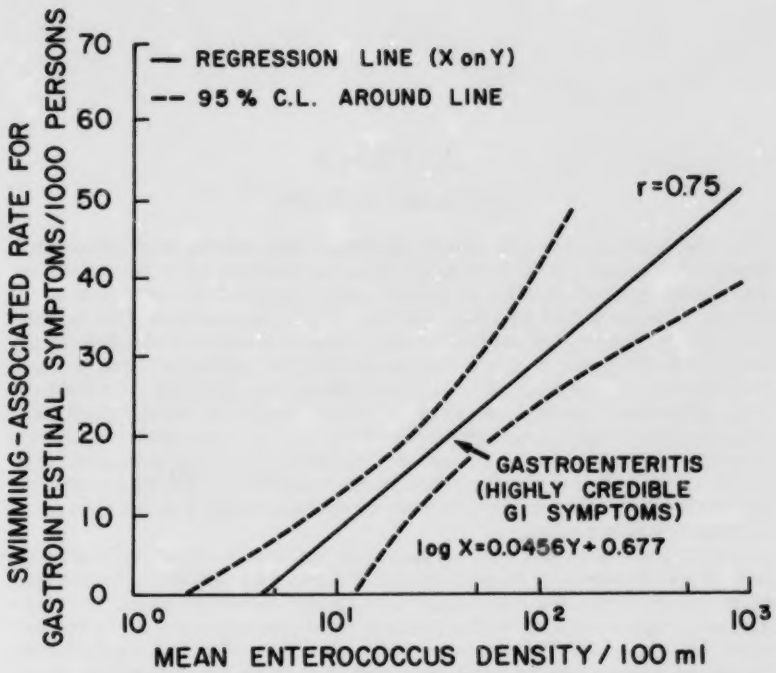
SECTION 1
INTRODUCTION

Existing health effects, water quality guidelines (often referred to as criteria) and standards for primary contact recreational waters, as recommended or promulgated by federal, state and local agencies, are generally stated as upper limits for fecal indicator densities. The current EPA guidelines state that, "Based on a minimum of five samples taken over a 30-day period, the fecal coliform bacterial level should not exceed a log mean of 200/100 ml, nor shall more than ten percent of the total samples taken during a 30-day period exceed 400/100 ml." Without exception, these guidelines suffer from two major deficiencies. The first is the paucity of epidemiological data which support some of them and the absence of any such support for others. At best, they relate to a "detectable risk" of infectious disease; at worst, they are based solely upon "attainment." The second, a consequence of the first, is that officials responsible for making decisions are given a "number," and this inherently limits the options available in decision making to compliance or noncompliance.

With the availability of a sufficient epidemiological base, a second option is available. In general terms, it is the promulgation of a criterion as defined herein; that is, a mathematically expressable relationship (model) of untoward effects among "users" to the quality of the water used. With reference to recreational waters, it is the relationship of the incidence or risk of disease among swimmers to the quality of the water as measured by the density of the infectious agent itself or an appropriate indicator. As shown herein, the major pollution-associated risk to recreationists is that of infectious disease consequent to swimming in waters polluted with human and, to a much lesser extent, lower animal fecal wastes. Therefore, the criterion relates infectious disease among "swimmers" to some measure of fecal pollution of the water. This approach then permits a decision as to "acceptable risk" based upon social, economic, medical, public health, and even political considerations (some form of cost-benefit or cost-effectiveness analysis). The acceptable risk of illness or its incidence can then be extrapolated from the criterion to yield a water quality limit (guideline), and the guideline can then be fixed in law to provide a standard.

This report presents such a criterion for marine recreational water quality, documents its epidemiological base, and discusses its applications and limitations. The recommended criterion shown in the figure below is the mathematical relationship (X on Y regression line) of the quality of the bathing water (X), as measured by the density of a specific fecal indicator (enterococci), to the incidence of swimming-associated gastroenteritis ("highly credible," gastrointestinal symptoms, Y). It is a deterministic model empirically derived from epidemiological and microbiological data obtained at multiple locations over several years. The deterministic form appears to lend itself more to cost-benefit types of analyses. The two input parameters to the model were not chosen arbitrarily. Rather, they were the output from an experimental design formulated to respond to the questions: Which are the "important" types of illnesses, and which is the "best" indicator? This is detailed in the body of the report.

This criterion is directed against potential human health effects consequent to the pollution of marine recreational waters with human fecal wastes, notably municipal sewage. It is a generalization which may not always hold true. Nevertheless, the fact that it has been found to be applicable at several locations has some implications concerning the ecology of the etiological agent(s) and the nature of the infectious process,



Recommended health effects criterion for marine recreation waters.

notably, the ubiquity of the agent in feces, sewage, and its receiving water. A similar criterion for freshwaters will probably be required, and the establishment of this criterion does not preclude the possible need for others, i.e., against the proliferation of aquatic organisms pathogenic for man (e.g., *Aeromonas hydrophila, Vibrio parahaemolyticus)* which respond to nutrient loading of the water.

The criterion may be used to develop guidelines for sewage treatment and outfall location. Knowledge of the transport and fate of both pathogens and indicator bacteria would provide a refinement for translating these target area criteria into effluent guidelines. It is hoped that the criteria will not be used to close swimming areas but rather to expand the available recreational resource.

Finally, when the study design for the EPA program was being developed in 1969-1970, it was thought that swimming in sewage-polluted waters would constitute a relatively minor route of transmission for gastrointestinal illness and that relatively high levels of pollution (as indexed by microbial indicator densities) would be required before gastrointestinal illness could be detected. These assumptions were made on the basis of existing notions and available information. Both these assumptions were incorrect. If the nonswimming rates for gastrointestinal symptomatology can be considered as those for the population at large, then it must be concluded that swimming in sewage-polluted waters constitutes a significant route of transmission for the illnesses obtained, at least for individuals of "swimming age."

SECTION 2
RECOMMENDATIONS

1. The health effects criterion for marine recreational waters presented herein should be considered for use by EPA since it is a relatively reliable generalization which is amenable to risk analysis, allows a wider choice of options at both the federal and local levels, and can be defended on the basis of epidemiological data.

2. A cost-benefit or cost-effectiveness type model should be developed for determining the acceptable risk or incidence of illness with regard to general and local factors.

3. Work should be continued toward the development of similar criteria for fresh recreational waters.

4. An intensive program should be initiated towards establishing the etiology of the gastroenteritis observed in these studies and developing methods for quantifying the agent(s) in environmental waters. This should be followed by a program to compare the biological decay of the agent(s) to its indicators under conditions best simulating those in open water.

5. The most resource responsible use of these criteria is their translation into effluent guidelines governing the design of sewage treatment facilities, the location of their outfalls and the decisions to be made relative to the degree of treatment and disinfection required. This and the preceding recommendation require the reinitiation of the program towards the development of realistic and facile methods for obtaining decay coefficients for indicators and pathogens on a case-by-case basis.

6. Nonspecific gastroenteritis is the major cause of outbreaks of disease from drinking water and shellfish consumption. The criteria suggest that there are measurable health effects associated with enterococcus or *E. coli* water densities as low as 10/100 ml via a route in which only 10-50 ml of water is ingested. Therefore, prospective epidemiological studies should be conducted as part of the reevaluation of existing standards for drinking water and shellfish-growing areas mandated by Sections 104(n)(1), 304 (a)(1) and 403(c)(1) of Public Law 92-500.

SECTION 3
BACKGROUND

Historically, the development of health effects, water quality guidelines and standards for recreational waters has followed a pattern characteristic of many such efforts to control pollution-associated health and ecological effects. The first step is the development of guidelines and standards dictated largely by *attainment* with the best available control technology. These are usually based upon limited epidemiological and ecological evidence and little, if any, data quantifying the risk in relation to the level of the pollutant in the environment. The second stage is the modification of these guidelines and standards on the basis of *detectable* risk using a limited quantity of data relating untoward effects to the environmental level of the pollutant. The last step in the process, the development of guidelines based upon *acceptable* risk, requires an epidemiological or ecological data base broad enough to mathematically model the relationship of some measure of water quality to the risk, degree or rate of untoward effects. With reference to health effects, water quality guidelines and standards for recreational waters, we have progressed through the second stage. This report will describe and substantiate criteria from which guidelines and standards based upon acceptable risk can be derived by risk analysis.

Sewerage systems for the disposal of domestic wastes from urban areas into nearby fresh and marine waters have been in existence in the United States since the turn of the century. By that time, it was clearly established that agents of enteric disease are excreted in large numbers in the feces of ill individuals and, hence, are potentially present in sewage and its receiving waters. A swimming-associated outbreak of typhoid fever was reported in 1921 (1). Yet, it was not until 1951 that Scott (2) proposed microbial guidelines for the quality of recreational waters; these were based solely upon attainment. It was 1968 (3) before guidelines related to detectable risks were recommended by the National Technical Advisory Committee (NTAC) to the Federal Water Pollution Control Administration (FWPCA). Criteria permitting the development of guidelines based upon acceptable risks are now available a decade later.

EXISTING GUIDELINES AND STANDARDS

As of 1972, the two guidelines or standards most commonly used by the various states and territories in the United States were a total coliform value of 1000/100 ml of water and a fecal coliform limit of 200/100 ml. The former appears to have developed from two sources, the anticipated risk of salmonellosis as obtained from calculations made by Streeter (4) on the incidence of *Salmonella* species in bathing waters and attainability as determined by Scott (2) from surveys conducted of Connecticut bathing waters. The Joint Committee of the American Public Health Association and the State Sanitary Engineers (5) adopted the Connecticut standard as did many of the state agencies.

The fecal coliform limits will be considered in more detail since, as can be seen from Table A1†, it is the most prevalent one used by the various states and it is the guideline currently recommended by the EPA (6). This guideline will be considered in terms of the data base which supports it, how it was derived, and the indicator system used.

The microbial guideline for primary contact recreational waters recommended by the

†When a table number is preceded by "A," the table is to be found in the Appendix.

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BACKGROUND

Historically, the development of health effects, water quality guidelines and standards for recreational waters has followed a pattern characteristic of many such efforts to control pollution-associated health and ecological effects. The first step is the development of guidelines and standards dictated largely by *attainment* with the best available control technology. These are usually based upon limited epidemiological and ecological evidence and little, if any, data quantifying the risk in relation to the level of the pollutant in the environment. The second stage is the modification of these guidelines and standards on the basis of *detectable* risk using a limited quantity of data relating untoward effects to the environmental level of the pollutant. The last step in the process, the development of guidelines based upon *acceptable* risk, requires an epidemiological or ecological data base broad enough to mathematically model the relationship of some measure of water quality to the risk, degree or rate of untoward effects. With reference to health effects, water quality guidelines and standards for recreational waters, we have progressed through the second stage. This report will describe and substantiate criteria from which guidelines and standards based upon acceptable risk can be derived by risk analysis.

Sewerage systems for the disposal of domestic wastes from urban areas into nearby fresh and marine waters have been in existence in the United States since the turn of the century. By that time, it was clearly established that agents of enteric disease are excreted in large numbers in the feces of ill individuals and, hence, are potentially present in sewage and its receiving waters. A swimming-associated outbreak of typhoid fever was reported in 1921 (1). Yet, it was not until 1951 that Scott (2) proposed microbial guidelines for the quality of recreational waters; these were based solely upon attainment. It was 1968 (3) before guidelines related to detectable risks were recommended by the National Technical Advisory Committee (NTAC) to the Federal Water Pollution Control Administration (FWPCA). Criteria permitting the development of guidelines based upon acceptable risks are now available a decade later.

EXISTING GUIDELINES AND STANDARDS

As of 1972, the two guidelines or standards most commonly used by the various states and territories in the United States were a total coliform value of 1000/100 ml of water and a fecal coliform limit of 200/100 ml. The former appears to have developed from two sources, the anticipated risk of salmonellosis as obtained from calculations made by Streeter (4) on the incidence of *Salmonella* species in bathing waters and attainability as determined by Scott (2) from surveys conducted of Connecticut bathing waters. The Joint Committee of the American Public Health Association and the State Sanitary Engineers (5) adopted the Connecticut standard as did many of the state agencies.

The fecal coliform limits will be considered in more detail since, as can be seen from Table A1†, it is the most prevalent one used by the various states and it is the guideline currently recommended by the EPA (6). This guideline will be considered in terms of the data base which supports it, how it was derived, and the indicator system used.

The microbial guideline for primary contact recreational waters recommended by the

†When a table number is preceded by "A," the table is to be found in the Appendix.

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EPA and adopted by most of the states (Table A1) is essentially that recommended by NTAC in 1968. Their recommendation was as follows:

> Fecal coliforms should be used as the indicator organism for evaluating the microbiological suitability of recreational waters. As determined by multiple-tube fermentation or membrane filter procedures and based on a minimum of not less than five samples taken over not more than a 30-day period, the fecal coliform content of primary contact recreational waters shall not exceed a log mean of 200/100 ml, nor shall more than 10 percent of total samples during any 30-day period exceed 400/100 ml.

Their rationale for specific limits was as follows:

> The studies at the Great Lakes (Mich.) and the Inland River (Ohio) showed an epidemiologically detectable health effect at levels of 2,300-2,400 coliforms per 100 ml. Later work on the stretch of the Ohio River where the study had been conducted indicated that the fecal coliforms represented 18 percent of the total coliforms. This would indicate that *detectable** health effects may occur at a fecal coliform level of about 400/100 ml; a factor of safety would indicate that the water quality should be better than that which would cause a health effect. . . .The Santee project correlated the prevalence of virus with fecal coliform concentrations following sewage treatment. Virus levels following secondary treatment can be expected to be 1 Plaque-Forming-Unit (PFU) per milliliter with a ratio of 1 virus particle per 10,000 fecal coliforms. A bathing water with 400 fecal coliforms per 100 ml could be expected to have 0.02 virus particles per 100 ml (1 virus particle per 5,000 ml).

The committee pointed out that the Public Health Service's three epidemiological studies on bathing water quality and health were the only base available for setting criteria, that these studies were far from definitive, and that they were conducted before the acceptance of the fecal coliform as a more realistic measure of a health hazard. The committee concluded that there is an urgent need for research to refine the correlation of various indicator organisms, including fecal coliforms, to waterborne disease.

Shortly after its publication, the NTAC guideline was attacked by Henderson (7) as being too restrictive. He set forth several arguments against the promulgation of microbiological standards on a nationwide basis; included were the broad confidence limits on the Most Probable Number (MPN) test (whether for total coliforms or fecal coliforms), temporal and geographic variability in pathogen to indicator levels, and the effect of differing sources of pollution (i.e., treatment plant effluents, stormwater run-off, farm lot wastes, etc.). However, the thrust of his attack was the paucity of defined epidemiological data in support of the NTAC guideline. To the contrary, he used the British experience (8); the observations from Santa Monica Bay, California (9); and the lack of morbidity or mortality data associated with swimming in support of a much less restrictive microbiological standard for bathing beaches, or even no standard at all.

In 1972, a panel of the National Academy of Sciences, National Academy of Engineering (10) came to the following conclusion:

> No specific recommendation is made concerning the presence or concentrations of microorganisms in bathing water because of the paucity of valid epidemiological data.

In explaining their inability to recommend a specific value they noted that many of the diseases that seem to be causally related to swimming and bathing in polluted waters are not enteric diseases or are not caused by enteric organisms. Hence, the presence of fecal coliform bacteria or of *Salmonella* sp. in recreational waters is less meaningful than in drinking water. Nevertheless, the substance of the NTAC guideline was adopted by the EPA in 1976 (6); and, by 1978, the large majority of the states and territories used it as a guideline or a standard (Table A1). Because of the seeming contradictions in the conclu-

*Author's emphasis.

sions drawn by different individuals from the same information, it is worthwhile to critically review that information.

DATA BASE IN SUPPORT OF EXISTING GUIDELINES AND STANDARDS

The data base in support of existing microbial guidelines can be sought from three different sources. These are (i) available morbidity and mortality statistics (including restrospective epidemiological analyses of case reports and disease outbreaks), (ii) output from predictive models, and (iii) the findings from prospective, controlled, epidemiological-microbiological studies.

Recreational Waterborne Outbreaks of Disease and Their Retrospective Analyses

Potentially, all the diseases which are spread by the anal-oral route and whose etiological agents are shed in the feces of ill individuals or carriers could be contracted by swimming in sewage-polluted water. This includes (i) bacterial diseases, such as salmonellosis (including typhoid and paratyphoid fevers), shigellosis (bacilliary dysentery), cholera, and gastroenteritis caused by enteropathogenic *E. coli*, *Yersinia enterocolitica*, etc., (ii) viral diseases such as infectious hepatitis, illnesses caused by enteroviruses (poliovirus, coxsackieviruses A and B, echoviruses, reoviruses and adenoviruses), and "nonspecific" gastroenteritis caused by the human rotavirus and parvo-like viruses, and (iii) diseases caused by a variety of protozoan and metazoan parasites, i.e., amoebic dysentery, giardiasis, ascariasis, etc.

In actuality, most of the reported outbreaks and cases of infectious disease in the United States associated with swimming in natural bathing places were nonenteric and included cases and outbreaks of otitis externa, swimmers' itch, leptospirosis, granulomas of the skin, and even very rare cases of tuberculosis and tularemia (11). The existing guidelines do not prevent these diseases. There have probably been less than 18 reported outbreaks of enteric disease, encompassing less than 700 cases, associated with swimming in sewage-polluted waters. Included are: four outbreaks of typhoid fever, three relatively small ones in the United States (1,12,13) and one of ten cases in Australia (14); an outbreak of shigellosis on the Mississippi River below Dubuque, Iowa (15); two very small and questionable outbreaks of enteroviral infections, one caused by Coxsackie A (16) and the other Coxsackie B (17); and an equally questionable outbreak of infectious hepatitis (18). The largest reported outbreak by far occurred in 1979; 187 individuals developed gastroenteritis within three days from swimming at two lakes within a park in Michigan during a three-day period in July (19).

Thus, it is understandable why workers such as Henderson (7) and Moore (8), after examining such reports, have questioned the need for water quality guidelines, much less standards, for recreational waters. There are, however, a number of considerations which suggest that case and outbreak reports by their very nature markedly understate the actual incidence of swimming-associated enteric disease. First of all, there are a number of other modes of transmission for these enteric diseases (i.e., drinking water, food, person-to-person contact) so that it is difficult to establish an association to a specific route. Second, much of the swimming occurs at beaches used on a daily basis or on weekends by urban and suburban populations who return to their homes each evening. This too adds to the difficulty of establishing a common source association with swimming at a given beach for "sporadic," geographically spaced cases of enteric disease. This is in contrast to drinking water where there is a geographic clustering of cases. It is of interest in this regard that the reported shigellosis and gastroenteritis outbreaks were detected under conditions where the population was geographically restricted, campers at state parks. Third, the levels of pollution at such beaches are relatively constant; thus,

one would not expect outbreaks (recognized because of temporal or spacial limits) but rather sporadic cases. Fourth, as will be pointed out later in this report, the immune status of the population to some of the potential etiologic agents will also tend to produce sporadic cases. Finally, the most commonly reported illness associated with drinking water and shellfish-associated outbreaks, a nonspecific gastroenteritis, is not a "reportable" disease. The usefulness of information from case and outbreak reports in developing criteria, guidelines, and standards is also limited because, with few exceptions and for obvious reasons, data on the quality of the water at the time of exposure are usually not available.

Prior to 1974, the only retrospective epidemiologic analysis concerning the risk of illness associated with swimming in sewage-polluted waters was carried out by Moore and his associates at some coastal communities along the coast of England and Wales (8). The basic design was to compare the incidence of swimming in a two-week period (for the ill individuals, it was the two weeks prior to the onset of illness) between two groups of individuals. The first was children ill with clinical poliomyelitis, and the second was a group of demographically paired controls (cohorts). Using this approach, Moore found no greater association of swimming among children ill with poliomyelitis than among their cohorts. In addition, he found very few cases of salmonellosis for which there was even the remotest association with swimming in polluted waters.

There were a number of problems with the experimental design used: (i) swimming was not defined rigorously; (ii) the time span between the actual swimming experience and the query as to its occurrence was protracted in many cases; (iii) it was difficult to establish a relationship to the quality of the water in which the individuals bathed; (iv) of necessity with this type of analysis in contrast to that used by Stevenson, there was a presumption as to which diseases were "important," poliomyelitis and salmonellosis; and (v) this type of analysis is rather insensitive except when conducted during an outbreak situation. In their report (8) Moore and his associates (the Committee on Bathing Beach Contamination of the Public Health Laboratory Service) noted some of these limitations and pointed out that, "A survey of this type could clearly not prove that poliomyelitis was never caused by bathing, and in any case such a presumptive finding might be contradicted by future events, but the results of the survey give no indication that further investigation along those lines is likely to be fruitful except in the negative sense recorded." Nevertheless, their findings do not warrant the conclusions drawn: that there is little, if any, risk of enteric disease from swimming in sewage-polluted waters unless aggregate fecal material is found therein and that aesthetic considerations will limit beach usage long before there is a significant risk of swimming-associated enteric disease. However, with regard to the two specific diseases in question, Moore's conclusions were probably correct since, even in the period subseguent to his report, there have been no outbreaks or cases of poliomyelitis shown to be asociated with the recreational use of water, and there has only been one outbreak of this disease even remotely associated with any of the waterborne routes (20).

There have been some cases of salmonellosis attributed to the recreational use of polluted waters, but, as Moore predicted, these have been associated with swimming in heavily polluted waters which were probably aesthetically unattractive. In the Australian outbreak, there was a broken sewage outfall (14); swimming in a sewage-contaminated drainage ditch (fecal coliform MPN 10^7/ 100 ml) was reported for the Alabama cases (13); the individuals in the Louisiana outbreak had been swimming in a river impacted by a broken sewer line (12); and four cases of typhoid fever detected in the Alexandria, Egypt bathing beach study to be described were all associated with swimming at a heavily polluted beach immediately impacted with raw sewage (21). The relatively few cases of swimming-associated salmonellosis which have been reported in the United States and the findings from those outbreaks are consistent with the high ID_{50}* for salmonellae (22), the decrease in *Salmonella* cases and carriers, and the increase in sewage treat-

*The number of microbial cells required to infect 50 percent of the exposed individuals.

ment. The removal of suspended solids during treatment decreases the number of multisalmonellae-containing particles. When the human ID_{50} data for salmonellae are considered, it would seem that such particulates would be required to produce swimming-associated disease, and the epidemiological setting for the above outbreaks are consistent with this hypothesis. Moreover, prior to 1979, the only outbreak of enteric disease unequivocally shown to be associated with swimming in sewage-polluted waters was a shigellosis outbreak on the Mississippi River below Dubuque, Iowa (15); and the ID_{50} for shigellae has been shown in volunteer studies to be several orders of magnitude less than that for salmonellae (22,23).

The information provided by the retrospective epidemiological analysis of the shigellosis outbreak (15) is of such importance in understanding the criteria which will be described that some detail is warranted (the equally important Michigan outbreak (19) will be discussed later in another context). Of 45 culture-positive cases studied, 43 (96 percent) of the individuals consulted a physician and 18 (40 percent) were hospitalized. Twenty-three individuals had a history of swimming in the area within three days of the onset of symptoms. Thirteen of them were swimming at a park area which, when sampled periodically during the month following the end of the outbreak, had a mean fecal coliform density of 17,500/100 ml *S. sonnei.* The same antibiogram and colicin type as the isolates from seven swimmers, also was recovered from these waters. A case-control analysis and a retrospective, cohort analysis of an additional 262 individuals revealed a statistically significant association of gastrointestinal illness with swimming but not with drinking well water or with food consumption. The illness was defined as diarrhea with fever or cramps occurring within three days. The rate among swimmers at the park was 12 percent. Of the swimmers, the highest attack rate and the best correlation to illness was among individuals who took water in their mouths and among children and adolescents (less than 20 years of age).

These findings must be used with caution since water quality measurements could be obtained only after the end of the outbreak and since the source(s) of the *Shigella* and indicator organisms in the water could not be unequivocally established. In addition, the data relate primarily to shigellosis, one of several swimming-associated diseases. Nevertheless, the report documents *a consequential outbreak of illness clearly associated with swimming in water polluted with fecal wastes*. More important, it would appear that the health effects occurred in the *absence of aesthetic deterioration sufficient to deter individuals from swimming in the area.* The concern with salmonellosis notwithstanding, this was a shigellosis outbreak, and the incidence of shigellosis in Dubuque had been steadily increasing over the four years prior to the outbreak.

Prospective Epidemiological Studies

Prior to 1973, the only prospective epidemiological studies dealing with recreational waterborne disease were those conducted by Stevenson and his associates in the 1950s (24). Since they were the basis for the NTAC and, hence, EPA guidelines and a point of departure for the studies to be described in this report, they will be described and analyzed in some detail. There were three studies. The first was conducted at two beaches on Lake Michigan in the vicinity of Chicago. The second examined illness rates among individuals at two locations, a swimming pool in Kentucky and a nearby stretch of polluted beach on the Ohio River. The third study was conducted at two marine beaches on Long Island Sound, one in New Rochelle, New York and other in Mamaroneck, New York. A calendar approach was used in all three studies, and this led to a number of problems with the experimental design. First of all, swimming was not defined rigorously enough so that any subsequent illnesses could be attributed exclusively to contact of the upper body orifices with polluted water as opposed to consumption of food at the beach, personal contact between beachgoers, aerosols potentially generated by toilet facilities, etc. Secondly because the trials were conducted over the entire summer, the

effects of day-to-day fluctuations in the pollution levels at the beaches were not eliminated. The consequence of this was that the mean indicator densities and, hence, the illness rates at the paired beaches in the first and third studies were not significantly different from each other. A third problem was that measurements were reported only for one indicator, total coliform bacteria.

In the first study, symptom rates among the beachgoers at the South Beach were no different than those at the North Beach. However, a statistically significant difference was obtained in the rate of total symptoms among individuals who were at South Beach during three "high" coliform density days as compared to those there during three "low" days. This was not true at the North Beach. The mean indicator density during the high days at the South Beach was 2300 total coliforms/100 ml. In the Ohio River study, the rate for total symptoms was higher among people at the chlorinated swimming pool than those at the polluted beach on the Ohio River. However, the age adjusted rate for gastrointestinal symptoms was higher for the individuals at the river beach than those at the swimming pool. The mean coliform density in the stretch of the Ohio River was 2700/100 ml. In the third study, conducted at the marine beaches in the vicinity of New York City, no differences in symptom rates could be obtained even when illness rates during "high" days and "low" days were compared.

Aside from those in the experimental design, there are a number of problems with the analyses of the data and the conclusions drawn thereof. First of all, Stevenson concluded that swimming per se resulted in a higher rate of illness; because of the experimental design, it can only be concluded that going to the beach results in a higher illness rate. Second, the comparison of illness rates for three high days versus three low days during the Lake Michigan study has been criticized in that the differences were shown for only one set of high versus low days,and no data are given for all the other possible combinations. Third, in the first study, the differences were reported for total symptoms, while in the second they were for gastrointestinal symptomatology; yet, both sets of data were used identically in the derivation of the NTAC guidelines. Because of the limitations in the experimental design and analysis, one could conclude the positive results were spurious and that there was no effect of swimming in sewage-polluted waters. Alternatively, the limitations in design and analysis notwithstanding, it might be argued that the findings described a reality obtained with a relatively insensitive epidemiological instrument.

There were also problems in the use of these findings in the derivation of the microbial water quality guidelines as set forth in the NTAC document. As noted earlier, there was no consistency in the type of symptom used in the derivation. Secondly, the authors of the NTAC document converted total coliform values into fecal coliform values in order to state the criteria in terms of "a more fecal specific indicator system." In fact, the lack of specificity in the total coliform values would be carried over into the fecal coliform guidelines in spite of the fact that the relationship between the two indicators was later determined on the same stretch of the Ohio River. Fourthly, it is now evident that the so-called fecal coliforms are not as fecal specific as was thought at the time that the NTAC guidelines were formulated. Finally, the findings from the Stevenson study and their use in deriving the NTAC and hence EPA guidelines are conceptually deficient in that they are not amenable to risk analysis. That is, they describe detectable not acceptable risks. Nevertheless, these were the best guidelines available, and, as noted by Shuval (25), target area guidelines are needed by engineers as the basis for the design of sewage treatment facilities.

Predictive Models

Predictive models based on pathogen densities in the water, the infective dose of the pathogens in question, and the relationship of pathogen to indicator densities have been equally unproductive in terms of producing the kinds of definitive information needed to support the existing guidelines. Attempts by Streeter (4), which were similar to those

used by Kehr and Butterfield (26,27) for other waterborne routes of transmission, assumed an ID_{50} for salmonellae of one, and this is several orders of magnitude less than those obtained later from human volunteer studies (22). A more recent study by Mechelas *et al.* (28) was equally unproductive, not because of the mathematical approach used but rather because of the poor quality of the input data to the model and the assumptions made as to which disease agents are important.

An attempt is made to justify the existing EPA guidelines from information on the relationship of fecal coliform densities to the frequency of *Salmonella* isolations in surface waters (6, 29). As pointed out elsewhere (30), this relationship has not been confirmed, especially when *Salmonella* densities rather than isolation frequencies are examined. Furthermore, it is conceptionally unsound to expect a consistent relationship between a fecal indicator and a pathogen which is not extremely prevalent in the population at large. Finally, considering the ID_{50} for salmonellae, a relationship to the frequency of its isolation hardly seems appropriate as a justification for a guideline.

In spite of the absence of epidemiological data showing swimming-associated cases of poliomyelitis, an attempt has been made to justify the guidelines based on some relatively poor data on poliovirus densities (including those of the vaccine strains) in the water, their relationship to fecal coliform densities, and the assumption that ID_{50} of poliomyelitis is one, if the virion is in the right place at the right time (31). This approach also is entirely unconvincing for the reasons stated earlier.

HEALTH EFFECTS RECREATIONAL WATER QUALITY INDICATORS

Ideally, recreational water quality indicators are microorganisms or chemicals whose densities in the water can be quantitatively related to potential health hazards resulting from recreational use therein. Historically, the concern has been with infectious enteric diseases, such as cholera and typhoid fever, whose etiological agents are excreted in feces and are spread by the contamination of water and food with fecal wastes.

There are a number of reasons why the pathogens themselves are not used for this purpose, and most of these are as valid today as they were at the turn of the century when the indicator concept was developed. First of all, as noted earlier, there is a wide variety of infectious agents potentially transmitted by the waterborne route, and, since the density of each will vary both temporally and spacially independent of the others, measurements would have to be made for each agent. Secondly, facile and reliable methods for quantifying most of the pathogens are unavailable, even today; in fact, there are no methods for quantifying what may be the most important (infectious hepatitis) and most prevalent (rotaviruses and parvo-like viruses) agents of enteric disease. Thirdly, and most important of all, because of the temporal variability in pathogen densities in feces and sewage (and hence their receiving waters), monitoring for the pathogens themselves is more akin to measuring the actual rather than the potential for disease. Thus, it is not surprising (i) that the indicator concept was developed shortly after fecal transmission of enteric pathogens was established, (ii) that the first three indicators suggested, *Escherichia coli*, *Streptococcus faecalis* and *Clostridium perfringens*, were fecal organisms (32), and (iii) that these, or groups to which they belong, are the three most commonly used indicators today (33,34). The regrettable fact is that, in each case, methodological rather than conceptual considerations led to the expansion of the group measured, i.e., coliforms and fecal coliforms instead of *E. coli*, fecal streptococci instead of *S. faecalis*, and spore-forming, sulfite-reducing anaerobes instead of *C. perfringens*. The health effects, water quality indicators which have been considered and the methods for their enumeration which have been developed under the EPA recreational water quality criteria program are presented in Table 1.

The coliform systems require some further discussion because they are the ones most

TABLE 1. METHODS DEVELOPMENT IN SUPPORT OF THE USEPA EPIDEMIOLOGICAL PROGRAM

Indicator	Method	Ref[1]	Indicator	Method	Ref[1]
Coliforms	mC	(35)	*P. aeruginosa*	mPA	(43)
E. coli	mTEC	(36)	*A. hydrophila*	mA	(44)
Klebsiella	mK	(37)	*V. parahaemolyticus*	mVP	(45)
Enterococci	mE	(38)	*Salmonella*	HVS	(46)
C. perfringens	mCP	(39)	Enteropathogenic *E. coli*	—[2]	(47)
Bifidobacteria	—[2]	(40)			
Coliphage	—[2]	(41)	Coprostanol	—[2]	(48)
C. albicans	—[2]	(42)			

[1] Literature citation for the method.
[2] No specific name.

commonly used and because most of the existing criteria are stated in terms of coliform or fecal coliform densities.

The total coliform population as commonly enumerated includes four genera in the family *Enterobacteriaceae: Escherichia, Klebsiella, Citrobacter,* and *Enterobacter*. It may also include other organisms, notably lactose positive members of the genus *Aeromonas* (49). Only *E. coli* is consistently and exclusively found in feces (50), although all five genera can be routinely recovered from domestic sewage in rather large numbers (51).

Belatedly, the total coliform system is being discarded for many applications because it is finally recognized that *Citrobacter* and *Enterobacter* species are not fecal specific. However, it is being replaced with the so-called "fecal coliforms," a group which includes thermotolerant *Klebsiella* as well as *E. coli* biotypes. There never was any evidence that the adjective "fecal" was properly applied. In fact, it has been known for some time that there are substantial extra-fecal sources of *Klebsiella,* (50, 52, 53), even for the thermotolerant biotype. In addition, *Klebsiella* is infrequently present in human feces, and then generally as a minor portion of the coliform population (50). A number of reasons have been given to justify the use of this coliform system instead of *E. coli*. It has been argued that much of the historical data is in terms of fecal coliforms, that the existing standards for recreational and shellfish waters are stated as fecal coliform densities, and that *Klebsiella* should be enumerated as a fecal indicator because it is an opportunistic pathogen. First of all, much of the historical data is in terms of total coliforms not fecal coliforms; secondly, the little epidemiological data in support of existing recreational or shellfish standards were developed in terms of total coliforms and extrapolated to fecal coliforms (3); thirdly, *Klebsiella* is an opportunistic pathogen of the respiratory and genito-urinary systems and not the gastrointestinal tract; finally, there are no data showing that *Klebsiella* infections have been obtained via the waterborne route, much less that they occur at environmental fecal coliform densities of less than 200/100 ml or 14/100 ml, the present EPA guidelines for recreational and shellfish-growing waters, respectively (6).

GUIDELINES BASED ON ACCEPTABLE RISK

Another problem with the existing microbial guidelines for direct contact recreational waters is that they are not amenable to, compatible with, or derived in the context of risk analysis. That is, the data from which the guidelines were derived and the manner of the derivation are related to detectable rather than acceptable risk. Therefore, decisions beyond acceptance or rejection of the specific limits cannot be made on the basis of scien-

tific, health, economic or sociological considerations This does not allow for deliberate decisions by local, state, or federal officials as to the costs to be paid for incremental decreases in the health risks involved. Finally, it presents a philosophical dilemma to individuals or groups who recommend guidelines based upon detectable risks. Once more sensitive epidemiological instruments are developed for measuring the risks involved or extrapolating them from existing information, they are forced to make the limits more restrictive in order to be conceptually consistent. In fact, this is precisely the position in which the EPA finds itself because of the results to be presented. The logical solution is to proceed to the next stage in the evolution of the guidelines, the use of those developed on the basis of acceptable risk.

The microbial water quality criteria for primary contact recreational waters to be recommended in this report and, hence, the guidelines and standards which can be derived from them are a radical departure from the guidelines currently recommended by the EPA and the guidelines and standards currently used by the various states. They differ conceptually from the existing guidelines (referred to as criteria) in that the usable information is presented in the form of dose-response type relationships rather than limiting microbial densities. Because the conceptual basis is different, it becomes important to define certain terms as they will be used throughout this document.

A health effects recreational water quality *criterion* developed for use with indicator systems is defined as a quantifiable relationship between the density of the indicator in the water and the potential human health risks involved in the water's recreational use. It is a set of facts or a relationship upon which a judgment can be made. A water quality *guideline* derived from the criterion is a suggested upper limit for the density of the indicator in the water which is associated with health risks which are considered unacceptable. The concept of acceptability implies that there are social, cultural, economic, and political as well as medical inputs to the derivation and that these may vary in time as well as space. A water quality *standard* obtained from the criterion is a guideline fixed by law. The relationship of guidelines to the criteria from which they are derived is shown graphically in Figure 1. Derivation of the guidelines from the criteria requires a

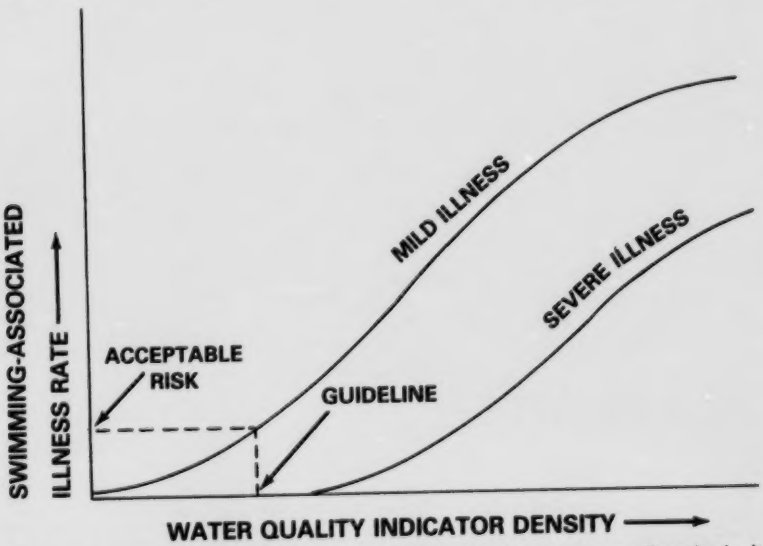

Figure 1. Graphic representation of the desired recreational water quality criteria. It is assumed that only an extremely small risk of "serious" illness will be accepted.

decision as to acceptable risk. This, in turn, is best obtained from some manner of cost-benefit or cost-effectiveness analysis which should include economic and sociologic considerations. Guidelines derived from such criteria differ from those currently in use in that they are consistent with risk analysis, allow for decision making, and are based on acceptable rather than detectable risks. This report presents such a criterion for marine recreational water quality, documents its epidemiological data base, and discusses its applications and limitations.

SECTION 4
STUDY DESIGN

The design of the epidemiological-microbiological program to develop health effects recreational water quality criteria was started in January 1969, shortly after the publication of the NTAC guidelines, and concluded in 1970. From the onset, the objective was to develop criteria amenable to risk analysis rather than guidelines based upon detectable risk (54). The experimental work was initiated in 1972 and concluded in 1978. A prospective approach similar to that used by Stevenson (24) was taken, in part to avoid prejudgements as to which diseases are spread by the recreational route, in part because a "nonspecific" gastroenteritis was the most common illness associated with the drinking water (55) and shellfish (56) routes of transmission, and in part because of Moore's (8) conclusion that further retrospective studies are unlikely to yield results other than those obtained in his study.

Marine beaches were chosen for the initial program because Stevenson's study at marine beaches did not produce demonstrable swimming-associated health effects, yet his freshwater findings were being applied to such beaches. Furthermore, if swimming-associated health effects were not obtained, this would tend to confirm the observed differences between fresh and saltwater beaches. If they were obtained, this would signal the need for a freshwater program, and the saltwater criteria could be used on an interim basis for freshwater beaches as well. The freshwater program was initiated in 1976. Finally, there were a number of heavily used and sewage-impacted marine beaches which could be studied along the Middle Atlantic and New England coasts.

PERCEIVED DEFICIENCIES IN STEVENSON DESIGN

An analysis of Stevenson's (24) study design, relative to the difficulties encountered and the results obtained, revealed several deficiencies which may have contributed to the inconclusiveness of his findings. To a large measure, they were due to the necessity of using the less expensive and time-consuming "calendar approach."

Definition of Swimming

Neither Stevenson, in defining his bathers as opposed to his nonbathing controls, nor Moore (8), in his inquiries concerning bathing, appears to have defined swimming such that individuals actually *at risk* — those whose upper body orifices were significantly in contact with the water — were isolated and examined. Thus, if swimming is not defined precisely, it is possible that differences in pollution-associated illness may be sought between two populations in both of which most of the individuals never were appreciably exposed. We considered this to be important from the assumption that less than 10 percent of the beachgoers would be classified as swimmers when immersion of the head in the water was used as the criterion for swimming. In fact, we were wrong. In almost every study, more than 60 percent of the beachgoers were classified as swimmers.

Multiple Exposures

The day-to-day variability in pollution levels requires that, ideally, the study group be limited to individuals who have had a single (one-day) swimming experience during the observation interval associated with a given trial. In both the freshwater (Lake Michi-

gan) and saltwater (Westchester) studies, the day-to-day variability as measured by coliform indicators was considerable; in fact, the range of indicator densities at each pair of beaches appreciably overlapped each other. Furthermore, in both these studies, the use of "calendars" to record illness made it necessary to limit the study to seashore residents. This maximized the probability that multiple exposures would occur. Stevenson, in comparing the incidence of illness during "high" and "low" pollution days, obviated only part of this difficulty.

Nonswimming Controls

Stevenson's nonswimming controls were individuals who did not go to the beach. Thereby, beach-going but not swimming-associated illnesses, such as gastroenteritis from improperly stored food, increased personal contact, use of communal toilets, etc., could be erroneously included in calculating the illness rates of the swimming as opposed to nonswimming populations. This could have affected illness-rate comparisons between "high" and "low" days as well as between beaches.

Demographic Considerations

Stevenson analyzed his data with consideration to age and sex but not to ethnic or socioeconomic (SES) factors. However, especially in the saltwater study, the test beaches appear to have been paired with reference to ethnic and SES factors of the resident populations. Susceptibility to disease, background rate of illness, nature of the swimming experience, and even the reliability of the respondents' information concerning illness and the swimming experience could vary by ethnic or social class.

Tidal Effects

Hourly variability in the pollution levels due to tide, wind, rainfall, etc., can present a problem in the interpretation of findings from epidemiological-microbiological trials. In Stevenson's study this was uncontrolled. Except in those instances where a "captive" study population is available, such as institutionalized individuals or organized groups, there is little that can be done to mediate such effects. Individuals at the beach during a given day can be expected to swim on several occasions during a half tidal cycle.

Indicators of Pollution

At the time of Stevenson's study, the state of the art was such that only two microbiological parameters were measured. Coliform determinations were made in accordance with confirmed test procedures described in the 13th Edition of "Standard Methods" (57). Enterococcus levels were also examined. These data were not used in the analysis because it was subsequently determined that, because of problems in assay methodology, the density estimates were too unreliable.

DESIGN CHARACTERISTICS

In response to the perceived deficiencies in the Stevenson studies, the calendar approach was not used (58). Rather, the participants were recruited at the beach and queried some 7-10 days later by phone or personal interview (mail questionnaires were tried and found to be unsatisfactory) concerning symptomatology which developed subsequent to the swimming experience. Other features of the design were as follows:

1. Only individuals whose upper body orifices were exposed to the water were classified as swimmers, and subjects were queried on the nature and duration of swimming activity. The validity of this information was pretested in the New York City study by observing family groups over a day at the beach and comparing these observations with information obtained at the day's end from a representative of the group. The more rigorous definition of swimming allowed for a beach-going

but nonswimming control group and thereby eliminated the bias from nonswimming associated illnesses.

2. Beach interviews were conducted only on weekends. Exposure was limited to a single day, or at most two successive days on a weekend. This was accomplished by eliminating individuals who swam in midweeks before and after the weekend trials from the study. The use of weekends maximized the size of the study population but limited the illness observation period to 8-10 days. This feature of the study facilitated the analysis of the data "by days," thereby obviating the effect of day-to-day variability in pollution levels. However, it eliminated from consideration illnesses with incubation periods exceeding nine days, notably infectious hepatitis (this was examined in the portion of the Egyptian study which dealt with Cairo visitors to the Alexandria beaches).
3. The impact of within-day variability in pollution, primarily attributable to tidal effects, could not be eliminated. However, in the first two years of the New York City study, an attempt was made to minimize this effect by choosing test and control beaches which were markedly different in the pollution levels reaching them. There also was an attempt to select trial dates when minimal tidal effects coincided with peak beach usage periods (usually 11 A.M. to 5 P.M.). This problem was potentially even more acute in the Boston Harbor study because of the greater tidal excursions and the unappealing nature of the intertidal zone. Because of this, swimmer and even bather densities were very low during low tides. Therefore, trials were conducted on those weekends when high or mid-tide corresponded to the hours of peak activity (11 A.M. to 5 P.M.). This forced the acceptance of lower mean indicator densities for this study.
4. Demographic effects, which could assert themselves as differences in susceptibility to infection, in swimming activity and in the reliability of respondent information, were minimized. This was done by selecting test and control beaches whose populations were demographically similar and by obtaining age, sex, ethnic, and SES information that could be used in isolating and identifying the influence of these factors.
5. The respondents were asked whether they remained home, remained in bed or sought medical advice because of the symptoms. This information was used to indicate disability.
6. In the pretest year of the New York City study, an attempt was made to validate the illness information provided by the respondents. This was done by providing the name of a physician in the reminder letter sent on the Monday following a trial and by requesting the names of other physicians consulted during the observation period. This was unsuccessful, and an alternative system was devised for validating gastrointestinal (GI) symptomology. Highly credible GI symptoms (HCGI): **were defined as (i) vomiting, (2) diarrhea with a fever or disabling enough for the individual to remain home, remain in bed or seek medical advice, or (iii) stomachache or nausea accompanied by a fever. The rates for HCGI symptoms were calculated and compared to those for total GI symptoms in order to determine if the trends were the same.**
7. The illness questionnaire solicited information on irritations and disturbances of the skin, upper respiratory tract, eyes, and ears. This was done not only against the possibility of pollution-associated infectious processes but also against that possibility of toxic and hypersensitive conditions attributable to chemical pollution and to pollution-associated changes in marine biota.

The sequence of events during and subsequent to the beach interview is shown in Table 2.

The experimental design as stated was generally followed for all the studies conducted. The notable exception was the Egyptian study and especially the portion dealing with health effects among Cairo visitors to the Alexandria beaches.

TABLE 2. SEQUENCE OF EVENTS FOR EPIDEMIOLOGICAL-MICROBIOLOGICAL TRIALS

Day of week	Day number	Activity	Function
Saturday	1	Beach interview, water sampling	(a) Obtain name, address, phone, etc. (b) Reject pre-trial midweek swimmers (c) Query on beach activity (d) Assay of water samples
Sunday	2	Same as Saturday	As above
Monday	3	Reminder letter	(a) Provide name of physician (b) Reminder to note illness
Monday	10	Phone or mail interview	(a) Obtain illness information (b) Reject post-trial midweek swimmers (c) Obtain remainder of demographic information.

INDICATOR ASSAYS

Water samples were collected in sterile bottles from just below the surface of the water, at "chest high depth," and periodically during the time when people were in the water. They were collected at 2-3 locations along the beach; and, in general, 3-4 samples were collected between the hours of 11 A.M.—5 P.M., the period of maximum swimming. The samples were "iced" and returned to the laboratory for assay within six hours of collection.

Assays of the water samples were performed to determine the densities of a number of potential microbial indicator systems. These are given in Table 1. Appropriate, evaluated methods were not available for bifidobacteria, coliphage, *Candida albicans*, and enterophathogenic *E. coli* or for the chemical, coprostanol, by the second year of the New York City study. Therefore, these indicators could not be included in the study. Membrane filter procedures were developed and used for most of the indicator systems examined. The methods are noted and referenced in Table 1. Membrane filter procedures were chosen because they provide more precise estimates than MPN determinations and allow larger samples to be examined than pour or streak plate procedures. A high volume (55.5 liters), MPN procedure (46) was used for *Salmonella, Klebsiella,* and *Enterobacter-Citrobacter*. Densities were determined by the mC procedure (35), although a method specifically for *Klebsiella* (37) was developed subsequent to the completion of the New York City study. In addition, fecal coliform densities were determined by the MPN procedure given in *Standard Methods for the Examination of Water and Wastewater* (57). Staphylococci were enumerated by a modification of the Chapman-Stone method for use in a membrane filter procedure (M. Levin, personal communication).

ANALYSIS OF THE DATA

Since the objective of the program was to relate the swimming-associated rates for symptoms, classes of symptoms or syndromes to some measure of the quality of the water, a temporal and spacial control population was provided. This was nonswimmers

(head not immersed in water) who were at the beach and, in general, came from the same family groups as the swimmers. Therefore, in most of the analyses, the swimming rate for a given symptom or group of symptoms was first compared to the nonswimming rate. Such differences were then examined relative to the pollution levels at different beaches or on different days or groups of days at the same beach. During the first two years of the New York City study, two beaches were used which, according to existing standards, varied widely with regard to their pollution levels. One was "barely acceptable" (BA) in that it was immediately adjacent to a beach posted as being unsafe for swimming; the other was "relatively unpolluted" (RU) according to existing guidelines and was at a much greater distance than the BA beach from any known pollution source. The choice of the beaches permitted making a decision as to "important" symptoms without recourse to a direct comparison with indicator densities. Chi-square analysis was used for this purpose.

The second premise of the program was that there would be no prejudgment as to which is the "best" indicator. Therefore, regression analyses of the geometric mean densities of each indicator against the symptom rates were used to determine which indicator provided the best correlation and, hence, was the best water quality indicator.

In the regression analyses, each point was defined by the symptom rate for a single trial (day), a cluster of trials with similar indicator densities or all the trials conducted over a given summer at a given location and by the corresponding geometric mean indicator density for all the samples collected at the beach. Regression analysis was also used to define the final criteria.

The studies conducted under the EPA program to develop recreational water quality criteria, the number of individuals from whom usable information was obtained, and the success rate for follow-up interview are presented in Table 3. The detailed findings from individual studies have been or will be presented in individual reports (21, 59-64).

TABLE 3. SUCCESS OF FOLLOW-UP PHONE INTERVIEWS AND THE NUMBER OF USABLE RESPONSES BY BEACH AND YEAR FOR STUDIES CONDUCTED UNDER THE EPA PROGRAM

Location	Beaches	% Follow-up During Study Year[1]			Number of Usable Responses During Year		
		1	2	3	1	2	3
New York City, NY	Coney Island	82.3	78.3	78.3	641	3146	6491
	Rockaway	86.6	82.9		681	4923	
Lake Pontchartrain, LA	Levee	77.2	77.9[2]		3432	2768	
	Fontainebleau					551	
Boston Harbor, MA	Revere	81.2			1824		
	Nahant	81.2			2229		
Alexandria, Egypt	Maamoura[3]	88.6	84.8	84.4	819	1492	1786
	—[4]		91.2	88.3		1696	2173
	Ibrahemia[3]	81.2	87.8	90.4	823	1117	2050
	—[4]		87.5	87.2		1159	1820
	Mandara[3]	82.9			1163		
	Sporting[3]		90.7	90.6		1257	2025
	—[4]		84.9	88.5		1243	2457

[1] Coney Island, Rockaway, 1973-1975; Levee, 1977-1978; Fontainebleau, 1978; Revere, Nahant, 1978; Maamoura, Ibrahemia, 1976-1978; Manara, 1976; Sporting, 1977-1978.
[2] Fontainebleau included with Levee.
[3] Alexandria residents.
[4] Cairo visitors.

SECTION 5
RESULTS OF THE STUDIES

NEW YORK CITY STUDY

This study was conducted in three phases (years) at Coney Island and Rockaway beaches selected with the assistance of the Bureau of Public Engineering, New York City Department of Health. The first phase, conducted in 1972 and 1973, was a pretest of the microbiological and epidemiological methodology and an evaluation of the suitability of the test beaches. In 1972, the reliability of information obtained from the interviewees concerning their bathing activities was examined using the method described earlier. Their responses were quite accurate regarding entrance into the water and immersion of the head therein. However, their perceptions as to how long they were in the water were less reliable, possibly because many of them bathed or swam on several occasions during the day. In 1973, trials were conducted at two beaches: the first, located between 18th and 22nd Streets on Coney Island, was designated as the BA beach; and the second, around 67th Street at the Rockaways, was designated as the RU beach (61).

The demographic distributions of the populations at the two beaches were similar (60); about two-thirds of the beachgoers were classified as "swimmers," and there were no striking differences between the Coney Island and the Rockaways populations with regard to the percentage so classified. Swimming was more frequent among males, Hispanic Americans, and the 0-19 years of age groups (Table A2). The differences in pollution levels as seen from the densities of a number of potential water quality indicators were markedly different (Table A3). The success rate for follow-up phone (not mail) interviews was acceptable (Table 3); however, an alternative to medical follow-up examination for validation of the respondents' information on symptomatology was required. The differential (swimming minus nonswimming) rates for the individual GI symptoms were generally greater at the Coney Island than at the Rockaway beach (Table A4), and statistically significant differences in the rates for GI symptomatology were obtained at the Coney Island but not the Rockaway beach (Table A5). The rate for respiratory symptoms was higher among swimmers than nonswimmers at the Rockaways, presumably due to the aerosolization of noninfectious material because of the heavy surf activity at the beach. Assays for *Salmonella* densities in the water were omitted from subsequent studies because of the low densities obtained (Table A6).

A detailed analysis of the second phase (1974) trials is presented elsewhere (60). The RU beach was changed from 67th Street to Riis Park at the Rockaways in order to increase the size of the study population. The consequence of this was a somewhat greater discrepancy between the BA and RU beaches with regard to ethnic and SES factors (60). With two exceptions, nearly all the 1973 findings were confirmed in 1974. They were the much lower mean indicator densities (Table A3) and the absence of differences between swimmers and nonswimmers for the individual respiratory symptoms or respiratory symptoms taken as a whole (Tables A4 and A5). Of the nonswimmers at the Coney Island and the Rockaway beaches, only 8.5 percent and 5.4 percent, respectively, did not go swimming because of existing symptoms or illness. None of the individuals at the BA beach and only 0.1 percent of those at the RU beach did not go swimming because of GI symptomatology.

Because of the larger study population, the rates for GI symptoms could be examined by demographic groups. However, the increase notwithstanding, data for disabling GI symptoms by type could not be analyzed statistically because of the small size of the resultant cells. The disabling GI symptom rate for swimmers was 10/1000 people higher than that for nonswimmers at the BA beach. At the RU beach, the rate for nonswimmers was higher than that for swimmers by 2/1000.

The results from the analysis of GI symptom rates by demographic groups for swimmers and nonswimmers at both beaches are presented in Table A7. The rates among children, Hispanic-Americans, and low-middle SES individuals who swam at Coney Island were significantly and appreciably higher than among those who did not. This was not so for the residual from each demographic category (adults, blacks plus whites, and the highest SES group). The GI symptom rate for nonswimmers among the children at the RU beach was appreciably higher than that for the corresponding group at the BA beach. The rate for nonswimming children at the RU beach was significantly higher than that for children who swam. This anomalous finding probably was not due to over-reporting, since this was also true of the "highly credible" portion. The nonswimming children may have been more prone to illness, although only 0.1 percent of these children or their respondents reported that they did not swim because of existing GI symptoms. The investigators favor the explanation that predominately white or black, higher SES children did not or were not allowed to swim because they were in the early stages of the illnesses for which they later reported symptoms (60).

Secondary transmission of illnesses within a family did not appear to provide an erroneous picture of the symptom rates associated with swimming (60).

The credibility of the information on gastrointestinal symptomatology was assessed by comparing the trends of all responses to those considered "highly credible." The rates for the "highly credible" symptoms among the four study groups were examined for the total population and separately for the children, Hispanic-Americans, and the low to middle SES groups. The trends for the highly credible portion were similar to those for all GI symptoms (Tables A5 and A7). Rates of HCGI symptoms for the three most sensitive groups of swimmers also were significantly higher than those for their nonswimming controls.

The finding of a statistically significant, swimming-associated rate of GI symtomatology at a BA but not at a RU beach showed that such effects could be determined and suggested that measurable health effects do occur even within existing guidelines and standards. However, these results did not speak to the overall objective of the EPA program, the development of criteria amenable to risk analysis as described earlier. The data from the third phase (1975) of the New York study along with the data obtained the previous two years were analyzed to further explore this possibility since a preliminary examination of the data from 1973 and 1974 suggested that criteria could be developed and that either *E. coli* or enterococci was the most appropriate indicator (61). Four beaches on Coney Island were studied in 1975. These were a "posted" area between 34th and 38th Streets and nonposted beaches between 18th and 24th Streets, 8th and 10th Streets, and 2nd and 4th Streets, Brighton.

As noted earlier, the data from the three years of the New York City study were examined by regression analysis in two ways. The first was by clusters of trials with similar mean indicator densities during a given summer. The second was by summers, that is, all the trials at a given beach during a given summer. Clustering was necessary in order to avoid data points with N values of less than 100 persons. This was accomplished with one exception, a N of 96 for nonswimmers in the analysis of *E. coli* densities. In a few instances, however, this was accomplished at the cost of grouping some trials with widely divergent densities. In almost all cases, this occurred with trials at the upper end of the density distribution for a given indicator. Where possible, "natural breaks" in the distribution of mean densities were utilized in clustering the trials. Nevertheless, this was somewhat arbitrary.

In both approaches, the attack rates for GI symptoms or the "highly credible" portion thereof (HCGI) were regressed against the mean indicator density. The log-linear regression equation:

$$Y = a \log X + b$$

was used in which X was the mean indicator density and Y the symptom rate.

The clustering of the trials for each of the indicators along with geometric mean density and range for each cluster is shown on Tables A8 through A18. The mean densities along with the data used in calculating the swimming-associated rates (swim-nonswim) of GI and HCGI symptoms for each cluster (some single trials were unavoidable) are shown for each indicator in Tables A19 through A29. The coorelation coefficients are presented in Table 4.

TABLE 4. CORRELATION COEFFICIENTS FOR TOTAL GASTROINTESTINAL SYMPTOMS AND THE "HIGHLY CREDIBLE" PORTION AGAINST THE MEAN INDICATOR DENSITIES FOR 1973-1975 TRIALS CONDUCTED AT NEW YORK CITY BEACHES

Indicator	Correlation Coefficients (r) for Highly Credible GI[1]		Gastrointestinal (GI)[2]		Number of Points (N)	
	Summ[3]	Clust[4]	Summ	Clust	Summ	Clust
Enterococci	.75	.96	.84	.81	8	9
E. coli	.52	.56	.56	.51	8	9
Klebsiella	.32	.61	.35	.47	8	11
Enterobact.-Citrobact.	.26	.64	.23	.54	8	13
Total coliforms	.19	.65	.12	.46	8	11
C. perfringens[5]	.19	.01	.38	−.36	5	8
P. aeruginosa	.19	.59	.25	.35	8	11
Fecal coliforms	−.01	.51	.01	.36	8	12
A. hydrophila	−.09	.60	−.08	.27	7	11
V. parahaemolyticus[5]	−.20	.42	.19	.05	5	7
Staphylococci[5]	−.23	.60	.71	.09	5	10

[1] Highly credible GI symptoms (see text for definitions).
[2] Total gastrointestinal (GI) symptoms.
[3] Analysis of data by summer by beach.
[4] Analysis of data by summer, by cluster of trials (days) with similar indicator densities.
[5] No data for 1973.

The mean densities and the ranges for each indicator for all the trials conducted during a given summer at a given beach are presented in Table A30. The corresponding data on GI symptom rates are given in Table A31, and the correlation coefficients for the regression of the swimming-associated rates on the mean densities in Table 4.

When the results from both approaches for examining the relationship of the indicator densities to GI symptoms (and especially the highly credible portion thereof) were considered, it was apparent that enterococcus densities provided the best correlation. Nevertheless, as planned, the two best-correlated indicators, enterococci and *E. coli*, were used in subsequent studies. It is of equal importance that total coliform and especially fecal coliform densities were less well correlated with gastrointestinal symptomatology.

The regression lines obtained for swimming-associated GI and HCGI symptoms against the mean *E. coli* and enterococcus densities when examined by summers and clusters of trials with similar indicator densities are presented in Figure 2.

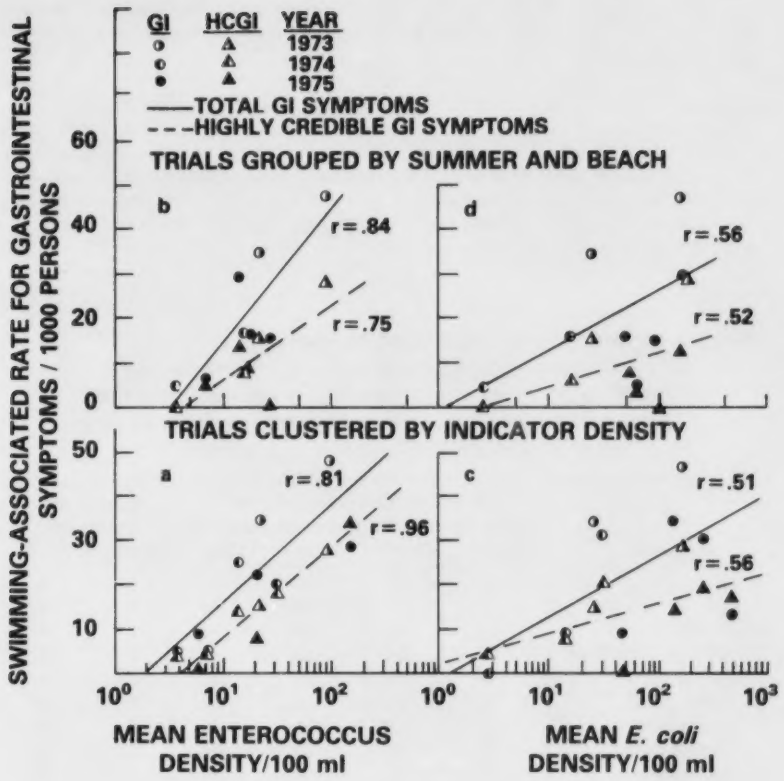

Figure 2. Swimming-associated (swimmer minus nonswimmer) gastrointestinal symptom rates against the mean enterococcus and *E. coli* densities in the bathing water for New York City study (1973-1975). Highly credible GI symptoms defined in text. In "a" and "c," trials clustered by similar indicator densities to yield points as shown. In "b" and "d," trials clustered by summer and beach. The actual trials clustered are given in Tables 8A through A31, Appendix A.

ALEXANDRIA, EGYPT STUDY

Animal infectivity studies conducted with most infectious agents yield sigmoid dose-response curves. At the inception of the EPA program, the relationship of illness among swimmers to indicator densities in the bathing waters was also expected to be sigmoid in nature. However, when the swimming-associated rates for GI symptoms were plotted in percentages on a scale that was not expanded to show differences (see Figure 3 as an example), the slopes of the lines were quite shallow relative to those seen in most dose-response curves. They may have represented the first parts of sigmoid curves, from which the expectation was accelerated increases in the symptom rates with further increases in the indicator densities at the beaches. An equally plausible explanation was that the regression lines obtained were the linear portions of basically sigmoid relationships (i) in which a measurable response was associated with the ingestion of very low

enterococcus or *E. coli* densities (note the Y axis intercepts in Figure 2) because of the differential survival of the indicators relative to the etiologic agent(s) over the travel time between the beaches and the sources of pollution, (ii) in which the shallow slopes of the regression lines were due to high levels of immunity to the infective agents(s) in the swimming populations, and (iii) from which the expectation was that the rates for the specific illness(es) involved would not accelerate with increasing levels of pollution as seen from the indicator densities.

Ideally, Figure 3 should be a log probability plot; practically, it makes no difference because of the low rates and relatively good "r" values obtained. Furthermore, since this is an indicator-illness rather than agent-response relationship, a log probability plot may not be appropriate.

It was thought that the nature of illness-indicator relationships obtained from studies conducted at beaches more heavily impacted with more immediate sources of raw sewage could be used to differentiate between the two possibilities. Therefore, an extensive search was made for beaches in the United States which not only met the above requirements but also were used by large numbers of individuals and were not posted as unsafe. No such beaches were found in the United States; however, several saltwater beaches which met these requirements were identified in Alexandria, Egypt and could be studied under the sponsorship of the PL480 program. Most of them were very heavily used during the summer, and, according to existing information, they varied in their pollution levels from some which were heavily polluted (even aesthetically undesirable) to some which were acceptable according to the EPA guidelines. The sources of pollution to the beaches were a number of short (about 50 meters) outfalls originally designed to accommodate overloading of the disposal systems due to rainfall. However, they now discharge sewage daily because the growth of the city created demands for sewage disposal which exceeded the capacity of the existing system.

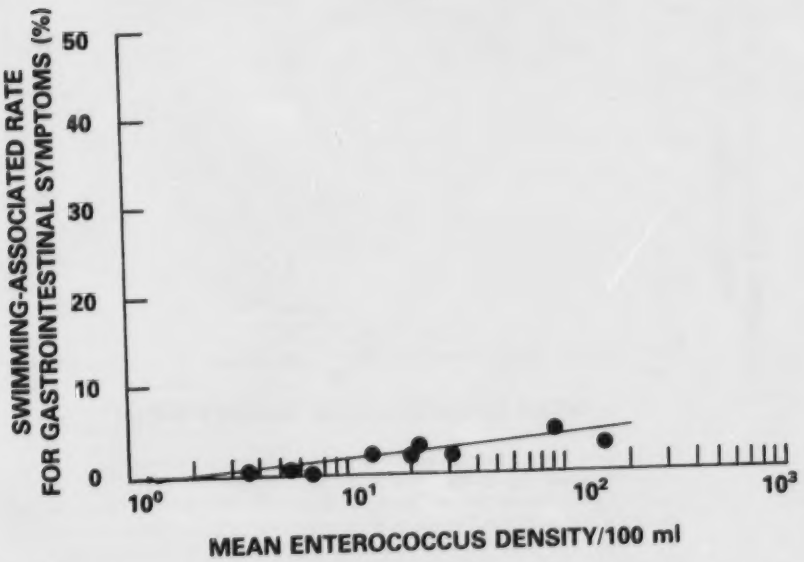

Figure 3. Data from Figure 2a shown as percentages on a scale more akin to that used in dose-response representations.

A preliminary survey of microbiological, demographic and user characteristics identified three beaches for the study — one very heavily polluted (Mandara), one moderately polluted (Ibrahemia), and one acceptable, but barely so according to the EPA guidelines (Maamoura).

The findings from the first year (pretest) of the study were similar to those obtained at the New York City beaches. Greater differences in the rates for vomiting and diarrhea among swimmers relative to nonswimmers were obtained at the heavily and moderately polluted beaches than at the acceptable one; and gastrointestinal symptomatology alone seemed to follow pollution as seen from *E. coli* and enterococcus densities, although the rates for most symptoms were higher for swimmers than nonswimmers at all three beaches. Children appeared to be the most susceptible portion of the population. However, a preliminary examination of the indicator-GI symptomatology relationship suggested an even shallower response curve than that obtained in the New York City study, this in spite of the higher pollution levels. Furthermore, there were indications that the GI symptom rates plateaued at mean *E. coli* and enterococcus densities of 200-300/100 ml (see data points for 1976 Alexandria residents in Figures 4 and 5). Finally, the *E. coli* and enterococcus densities associated with a "detectable" illness response (X axis intercepts) were higher than those obtained in the New York City study; those for enterococci were higher than those for *E. coli,* the indicator with the poorer survival characteristics in saltwater (65). These findings recommended the second hypothesis noted earlier in this section.

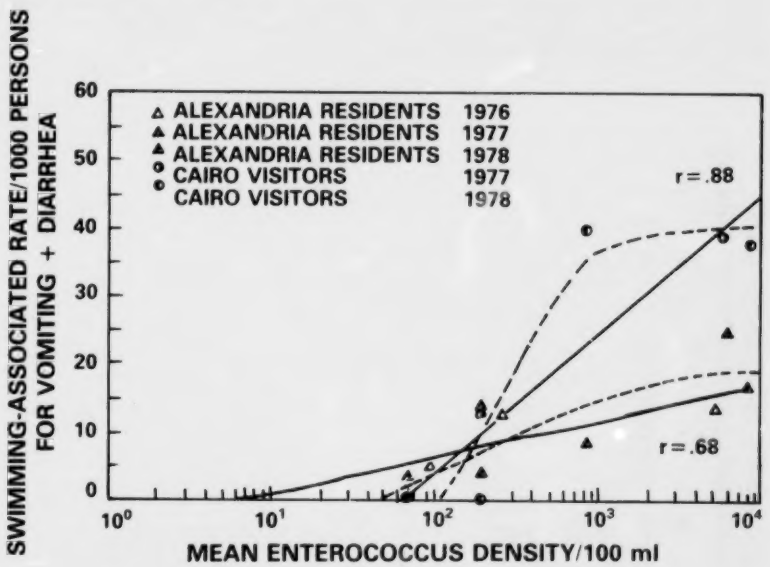

Figure 4. Swimming-associated rates for vomiting or diarrhea against the mean enterococcus density in the water (Egyptian study). The correlation coefficients (r) are those for the linear relationship. The dotted lines are the author's interpretation of the overall relationship from those seen for the individual years. Data given in Table A38, Appendix A.

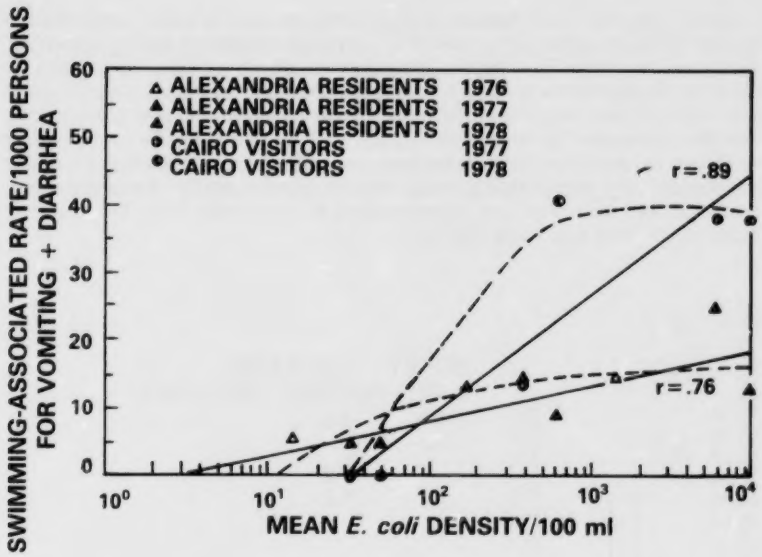

Figure 5. Swimming-associated rates for vomiting or diarrhea against the mean *E. coli* density in the water (Egyptian study). See Figure 4 caption for explanations.

Because of the above findings, the study was not only continued but extended to examine Cairo tourists at the Alexandria beaches as a population which, with regard to its immune status, might be more akin to that in the New York City study. In addition, the follow-up period with the Cairo population was extended to consider infectious hepatitis which, along with typhoid fever, is much more prevalent in Egypt than in the United States. This required a somewhat altered experimental design. The "Cairo visitors" were recruited at the beach shortly after their arrival in Alexandria. Follow-up inquiries were made in Alexandria and, as required, in Cairo at weekly intervals over a 30-35 day observation period. Follow-up in Alexandria was facilitated because most of the tourists remained in Alexandria for 2-4 weeks in rented cabanas at the beach. The altered design with the Cairo visitors precluded the use of "weekend trials" and, therefore, made the results more subject to the vagaries of day-to-day variability in pollution levels. However, the levels were relatively constant since there was little rainfall during the summer and the sewage impacting these beaches was untreated.

The pumping schedule at the Mandara outfall was changed in 1977, presumably because of the 1976 findings; this was reflected in the lower *E. coli* and enterococcus levels obtained at this beach in the spring of 1977. Because of this, "Sporting" was substituted for Mandara as the heavily polluted beach in the 1977 and 1978 trials.

The swimming and nonswimming rates for the various symptoms among the Alexandria residents and the Cairo visitors for each of the three years of the study are given in Tables A32 through A34. The swimming-associated (swimmer minus nonswimmer) rates are summarized in Table A35. Only data from the first weekly follow-up with the Cairo visitors were used in the analyses of the 1977 findings in order to maintain comparability with the data obtained for the Alexandria residents. For the same reason, the symptom rates given for the Cairo visitors in 1978 are those for individuals who swam

1-2 days during the week. Because of the resulting decrease in usable responses and because of the disparity in the rates of GI and upper respiratory tract symptoms for nonswimmers obtained from the first as compared to the second follow-up inquiry (Table A36), the data for the first two follow-up inquiries were used to calculate the symptom rates for Cairo visitors in the 1978 trials. It can be seen from Table A35 that, with only three exceptions, the rates for the various symptoms were higher for swimmers than nonswimmers. However, only with the gastrointestinal symptoms (vomiting or diarrhea) and possibly fever did the rates generally increase with the pollution levels at the three beaches as seen from the *E. coli* or enterococcus densities (Table A35). The rates were higher for children than adults (Figure 6).

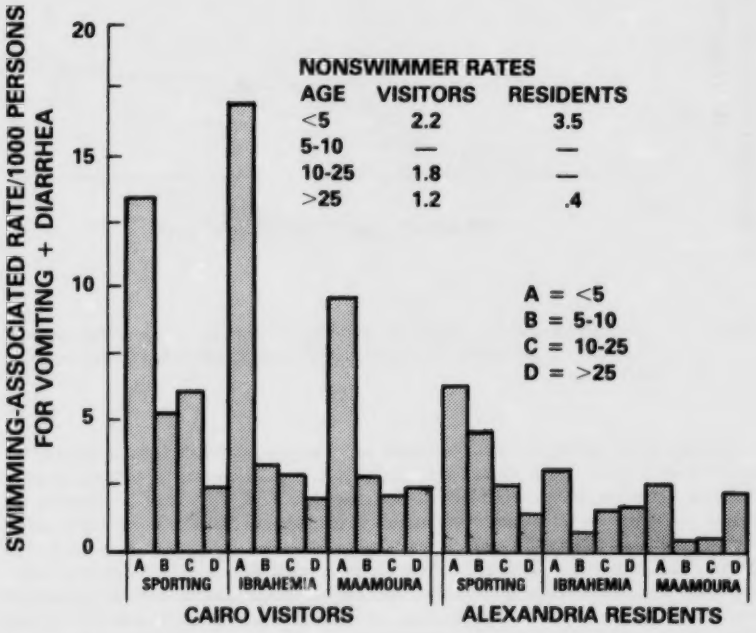

Figure 6. Age-specific, swimming-associated rates for vomiting or diarrhea by beach and study population for the 1977 Egyptian trials. The age-specific rates for the nonswimming Alexandria residents and Cairo visitors are shown as an insert.

Another finding that paralleled one obtained in the New York City study was that, with the exception of GI symptoms (vomiting or diarrhea) at the least polluted beach (Maamoura) and ear complaints at the other three beaches, the swimming-associated symptom rates per 1000 person-days decreased with increasing swimming activity. This can be seen from the analysis of the 1978 data from the Cairo visitors by the number of swimming days per week (Table A37).

Only three cases of jaundice were detected among the Cairo visitors, and there was no association to swimming, much less swimming in polluted waters. Four cases of typhoid

fever were found among swimmers at Sporting, the most heavily polluted beach.

The regression lines for the swimming-associated rates for vomiting or diarrhea against the enterococcus and *E. coli* mean densities for the Alexandria residents and Cairo visitors are shown in Figures 4 and 5. The data from which the lines were drawn are given in Table A38. As expected, the slopes of the lines for the Cairo visitors were greater than those for the Alexandria residents. Straight lines could be fitted to these illness-indicator relationships for the data from both the Cairo visitors and the Alexandria residents. However, examination of the relationships for the individual years suggests that there are plateaus as shown.

The plateaus, the differences in the indicator-illness curves for the Cairo visitors as compared to the Alexandria residents, and the higher GI symptom rates for children as compared to adults support the premise that the swimming populations were largely immune to the etiological agent(s). Moreover, from the similarities in the symptomatology and age distributions of symptoms in the Egyptian and New York City studies and the differences in the slopes and intercepts on the Y axis of the indicator-illness curves, we recommend the second explanation for the relationships obtained in the New York City study. However, these predictions relate only to the specific agent(s) responsible for the observed GI symptomatology. Swimming-associated illness rates exceeding those predicted by the illness-indicator relationships obtained from the New York City and Egyptian studies could occur with etiologic agents to which there is little immunity in the population . Thus, an attack rate of 13 percent appeared to be associated with fecal coliform densities of about 17,500/100 ml in the Dubuque shigellosis outbreak (15).

In addition to providing insights into the widespread distribution of the swimming-associated, pollution-associated gastroenteritis, its etiology and the role of immunity, the results of the Egyptian study suggest the circumstances under which typhoid fever could become a problem via the recreational route, i.e., near an outfall for untreated sewage. This finding, along with the available ID_{50} data for these agents (22), suggests the importance of the removal of particulates during primary and secondary sewage treatment in preventing the recreational transmission of this disease and other diseases whose agents have high infective doses. The absence of swimming-associated infectious hepatitis in an area where the endemic rate is high would suggest that, by the time they start to swim, even the Cairo children have been exposed and are immune to infection with hepatitis A virus.

LAKE PONTCHARTRAIN STUDY

This study was conducted during the summers of 1977 and 1978 at Levee beach which is located near the "mouth" of Bayou St. John on Lake Pontchartrain. Individuals swam both in the mouth of the Bayou and in a nearby roped-off area. In 1978, a second beach (Fontainebleau) located across the Lake was also included. The setting for the study differed in a number of important ways from that for the New York City study; there is very little tidal activity; the water is brackish (about 5 percent) and warmer during the summer; there is no beach as such but rather a series of steps leading downward from the grassy bank into the water. Most important of all, the sources of pollution were much less defined. According to local authorities, there were no discharges of sewage wastes into the Lake or Bayou St. John. However, high coliform densities were observed at the beach following rainfalls during "wet years." Presumably these were due to stormwater discharges reaching the beach via canals and bayous which empty into the Lake west of the beach.

Because of the ill-defined pollution sources, there was some reluctance to conduct a study at this location. However, the findings from sampling conducted in 1976 confirmed the high indicator densities following rainfalls and revealed moderate enterococcus densities during dry weather. Because of this, because of the desire to test the illness-indicator relationships under a different set of environmental conditions and be-

cause this study could be a vehicle for separating the two indicators which emerged as the best ones from the New York City study, trials were conducted in the summer of 1977.

When the rates for the individual symptoms were compared for swimmers versus nonswimmers, statistically significant differences were obtained only for vomiting, diarrhea, stomachache, earache, and skin complaints (Table A39). When the symptoms were grouped into categories, significantly higher rates for swimmers were obtained only for GI and "highly credible" GI symptoms, although there were differences for all the categories (Table A40). In general the GI symptom rates were higher for children than adults (Table A41). There were several striking aspects of the findings which suggested (i) that the major source of the infective agents was in the Bayou and not stormwater runoff arriving from west of the beach, (ii) that enterococcus densities were better correlated with the GI symptom rates, and (iii) that, because of this, the source of the pathogens was rather remote (in time) from the beach.

First of all, the mean enterococcus densities in the "mouth" of the Bayou were generally higher, and at times markedly so, than those at the beach (roped-off area); this was much less true of *E. coli* (Table A42). Secondly, in contrast the findings from the New York City and Egyptian studies wherein the *E. coli* and enterococcus densities tended to parallel each other, high *E. coli* densities were associated with low enterococcus levels and vice versa. The former occurred during the period 7/30-8/28 when the average daily rainfall exceeded 0.43 inches per day. The overall swimming-associated GI symptom rates for the trials conducted during this period were less than those for the trials conducted prior to July 30 when the average daily rainfall was 0.12 inches per day and the enterococcus densities exceeded those of *E. coli* (Table A43). Thirdly, the indicator densities in the roped-off area approached those in the Bayou only during the rainy period and then only for *E. coli* (Table A43). Moreover, the lower enterococcus densities and GI symptom rates during the "wet period" suggested that stormwater reaching the beach from the west reduced the pathogen and enterococcus densities at the beach by dilution or exclusion of organisms whose source presumably was in the Bayou. Fourthly, the trials during which there were high rates of swimming-associated GI symptoms corresponded better with high enterococcus than high *E. coli* densities (Table A44); in fact, when the swimming-associated GI and HCGI symptom rates for the four lowest *E. coli* days were compared to those for the four highest days, the former were higher than the latter (Table A45). Finally, it has been reported (65) that enterococci survive better than *E. coli*, especially in salt water.

The input data to the criteria model are given in Table A44. The considerable trial-to-trial variability in the indicator densities required that, even for the regression analysis by summers, the trials be clustered according to their indicator densities.

The findings from the 1978 trials differed from those obtained in 1977 in a number of ways, and some of the differences made the interpretation of the illness-indicator density data even more difficult: 1978 was a somewhat "drier" year than 1977, and, in general, the densities of both indicators were reduced. Nevertheless, the swimming-associated rate for GI symptoms was almost the same (39/1000 persons in 1978 as opposed to 42/1000 in 1977). This suggested that rainfall induced stormwater runoff to the beach (and the resulting elevated indicator densities) was not the source of the infective agents responsible for the observed symptomatology.

The rationale derived from the examination of the 1977 information was applied to the 1978 data as follows. It was assumed: (i) that, during a "relatively dry" year, the travel time down the Bayou was even more protracted, (ii) that, because of this, even the enterococcus densities were reduced relative to the pathogens, and (iii) that these lower enterococcus densities would be masked at the beach and even at the Bayou by those carried in with the stormwater. Three trials were associated with especially high *E. coli* and enterococcus densities in which the levels at the beach were as high or higher than those in Bayou (Table A46). Because these were the same three days during which there was

a half-inch or more rainfall (Table A46), the data from these three trials were eliminated from the analysis. Since the premise was that the source of the infective agents was the Bayou and since the roped-off area was expected to be more heavily impacted by stormwater, the remaining trials were grouped into high and low days based upon the Bayou indicator densities (Table A46), and these were used to calculate the mean indicator densities to which the symptomatology rates were compared. The mean indicator densities and associated GI and HCGI symptom rates as used later in the development of the criteria are given in Table A47.

The data from Fontainebleau beach, because of the relatively little trial-to-trial variability in the indicator densities, were used to derive a single relationship.

The 1978 data differed from the 1977 data in yet another way. In 1978 there were also statistically significant differences between swimmers and nonswimmers for the respiratory, other, EEN (ear, eye and nose) as well as disabling GI symptoms. This may have reflected a change in the pathogens present.

The Lake Pontchartrain study achieved its major objective. It, along with the third year of the New York City study, clearly showed enterococci to be superior to *E. coli* as a recreational water quality indicator. In addition, there were some important implications of the results obtained. First, they suggested some conditions under which even the enterococci may be deficient as a recreational water quality indicator. Second, they suggested that the etiological agent(s) of the swimming-associated gastroenteritis survives transport in the aquatic environment extremely well. Third, they provided a reasonably clear indication that stormwater runoff is less hazardous than wastewater discharges, and, because the two indicators are not specific for human fecal wastes, they may overstate the risk under these conditions.

BOSTON HARBOR STUDY

This study was conducted at two beaches in Boston Harbor in 1978. Its objective was to expand the data base for the criteria being developed and to confirm the observation that the measurable swimming-associated health effects were obtained at strikingly low indicator densities. As in the Lake Ponchartrain study, the sources of pollution to the two beaches, Revere and Nahant, were not as well defined as those in the New York City or Alexandria, Egypt studies. At the time it was screened for suitability in 1978, the mean enterococcus and *E. coli* densities at Revere beach were about 80/100 ml and exceeded those at Nahant by about an order of magnitude.

Four trials were conducted at each beach during June and July of 1978. The rates for the symptom categories are presented in Table A48. At both the Revere and Nahant beaches, the highest swimming-associated rates were for the total and HCGI symptoms, although the differences between the swimmer and nonswimmer rates were not significantly different. The differential rates were consistently greater at Revere than at Nahant beach, even though the mean indicator densities at the two beaches were not appreciably different (Table A49). This observation underscores the fact that the relationships being derived are generalities which may vary somewhat with a number of factors in the swimming population (i.e., their immune status, background illness rates, and even in the temporal and spacial relationship of the beach to its source of pollution). Nevertheless, the swimmer rates for GI symptoms were consistently higher than those for nonswimmers even at rather low levels of pollution as seen by the enterococcus or *E. coli* densities. The mean enterococcus and *E. coli* densities at Revere beach were less than those observed the previous year. These and the corresponding rates for GI and HCGI symptoms calculated by summer and by clusters of trials are given in Tables A49 and A50. The relationship of the swimming-associated rates to the indicator densities were more akin to those obtained at Lake Pontchartrain rather than New York City, i.e., higher rates for given indicator levels. This suggests differential biological decay of the indicators relative to the pathogens over more protracted transport times between the sources of pollution and the impacted beaches.

SECTION 6
DEVELOPMENT OF CRITERIA

In order to reach the objective of the overall program, the development of health effects criteria for marine recreational waters, four questions needed to be answered. They were:

1. *Does swimming in sea water per se carry with it an increased risk of illness and, if so, to what type?*

Stevenson's findings (24) suggested that it is so for fresh, but not sea, waters Those from the EPA program indicated this was true of sea water swimming as well. In the Stevenson study, it was observed most with ear, eye and nose complaints, less so with upper respiratory symptoms and least with gastrointestinal symptomatology.

2. *Is there an association of the illness rates to pollution from domestic sewage; and if so, to what type of illness?*

Stevenson's results (24) suggested there is such an association for swimming in freshwater but not in seawater. His results were equivocal as to the type of symptom. Moore (8) could find no association for poliomyelitis or salmonellosis. The conclusion from the EPA program is unequivocal; there is an increased risk of gastroenteritis associated with swimming in waters more as opposed to less polluted with sewage. Furthermore, the increased risk occurs at beaches which meet and even exceed the existing EPA guidelines and those of most of the states. Both the Egyptian and American studies suggest that fever often accompanies the GI symptoms. There were no indications in any of the American studies that anyone required hospitalization.

With the Cairo visitors to the Alexandria beaches, no association between swimming and infectious hepatitis (IH) could be detected, even among individuals who swam in waters so heavily polluted that they were aesthetically undesirable. The assumption was that the children of the Cairo visitors, coming from better sanitary environments and swimming in waters receiving waste loads from a population with a high endemic rate of IH, would be the most susceptible portion of the swimming population. However, even these children may have been exposed and rendered immune to the agents by the age they start swimming (immersion of the head in the water). A different study population is needed to resolve this question.

It is of interest that four cases of typhoid fever did occur among swimmers at the heavily polluted, aesthetically undesirable beach. This was not statistically significant and may have been a spurious result. However, since the ID_{50} for salmonellae is high (22), and that for IH is thought to be fairly low, these results lend credence to the postulated immunity explanation for the absence of IH among swimmers. There was no indication of poliomyelitis in any of the studies. Thus, Moore's conclusions (8) with regard to poliomyelitis and salmonellosis remain as true today as they were then.

3. *Which, if any, of the potential indicators of water quality best defines the association of GI symptomatology to water quality?*

The New York City study was designed to answer this question for beaches impacted with the sewage effluents from large urban areas. The Coney Island beaches were affected primarily by sewage emerging from the mouth of the Hudson River, and al-

though these were combined effluents subject to the effect of rainfall, treated to various degrees, and chlorinated only in part, they nevertheless represented a *relatively* well defined source. The criterion used to select the "best" indicator was the degree of association between its levels in the bathing water and the swimming-associated rate for gastrointestinal symptoms. It was evident from the New York City study that enterococci and, to a *much lesser extent*, *E. coli* were the best indicators of those examined (Table 4). *Fecal coliforms were a relatively poor indicator system.*

The marked superiority of enterococci over *E. coli* as a recreational water quality indicator was confirmed in the subsequent studies conducted in the United States. Higher correlation coefficients (r) for the mean indicator densities in the water against the swimming-associated rates for total or highly credible GI symptoms were obtained with enterococci than with *E. coli* (Table 5). However, comparable correlation coefficients were obtained for the two indicators in the Egyptian studies (Table 5). One explanation for this difference lies in the nature and proximity of the pollution sources. The sources of fecal pollution to the Alexandria beaches were untreated, not disinfected, and relatively close to the beaches. A portion of those to the New York City beaches were both treated and disinfected, and they were more distant from the beaches. Furthermore, more of the sewage emerging from the Hudson River and Upper Hudson Bay was treated and/or disinfected in 1975 than in 1974. This appears to correspond with poorer correlations of the indicator densities to gastrointestinal symptomatology, especially for *E. coli* (compare the 1973-74 to 1973-75 "r" values in Table 5). Insofar as could be determined, there were no nearby sources of human fecal wastes to either the Lake Pontchartrain or Boston Harbor beaches.

Implicit to the above explanation is the conclusion that enterococci more closely resembles the pathogen(s) than does *E. coli* with regard to its survival characteristics during sewage treatment, disinfection, and transport in the marine environment. Furthermore, as the level of sewage treatment and disinfection increases and/or the transport time becomes more protracted, even the densities of the enterococcus indicator are not maintained comparable to those of the pathogen. This and other considerations to be discussed notwithstanding, the mean enterococcus density does provide a meaningful and useful index of the potential for the observed gastrointestinal symptomatology.

Four possible indicator systems were not evaluated in the course of the New York City studies. As part of the EPA program, new methods have been developed or existing methods have been modified for each of the four indicators, *Candida albicans* (42), bifidobacteria (40), coprostanol (48) and male specific DNA, coliphage (41). Some preliminary evaluations were made with the first two. The densities of *C. albicans* were too low and variable in sewage-polluted waters to be of much value. Bifids were found to be fecal specific and reasonably human specific; however, their use as the basis for the criteria is precluded by their exceedingly poor survival during chlorination and transport in aquatic environments. Nevertheless, the recovery of these bacteria from environmental water samples indicates an "immediate" source of undisinfected human or, to a lesser extent, porcine fecal wastes (40). Coprostanol and the f-1 male specific coliphage need to be evaluated as water quality indicators and as conservative tracers.

4. *Can the relationship of swimming-associated health effects to the quality of the water, as determined by a microbial or chemical indicator, be quantified sufficiently to produce health effects quality criteria for marine recreational waters?*

The response to this question will be considered in the next section.

The Criteria

The regression lines for the rates of swimming-associated GI and HCGI symptoms against the mean enterococcus and *E. coli* densities when examined by trials clustered by indicator density or by summer are presented in Figure 7. The input data for the analyses

TABLE 5. CORRELATION COEFFICIENTS FOR ENTEROCOCCUS AND *E. coli* DENSITIES AGAINST THE GASTROINTESTINAL SYMPTOM RATES FOR UNITED STATES AND EGYPTIAN STUDIES

Symptom	Studies	Years	Correlation Coefficients (r) for trial clustered by: Indicator Densities[2]		Summers[3]	
			Enterococcus	*E. coli*	Enterococcus	*E. coli*
Gastrointestinal	New York City	1973-74	.90	.94	.95	.96
	New York City	1973-75	.81	.51	.84	.56
	L. Point.-Boston Harbor[1]	1977-78	.84	.16	.86	.02
	All U.S.	1973-78	.82	.25	.86	.20
Highly Credible GI	New York City	1973-74	.98	.96	.96	.97
	New York City	1973-75	.96	.56	.75	.52
	L. Point.-Boston Harbor	1977-78	.62	.57	.74	.54
	All U.S.	1973-78	.75	.54	.72	.52
	Alex., Egypt (Resid.)[4]				.69	.76
	Alex., Egypt (Visit.)[5]				.88	.87

[1] Lake Pontchartrain and Boston Harbor studies analyzed together.
[2] Trials clustered by similar indicator densities.
[3] Trials grouped by summers.
[4] Alexandria residents at Alexandria beaches.
[5] Cairo visitors to Alexandria beaches.

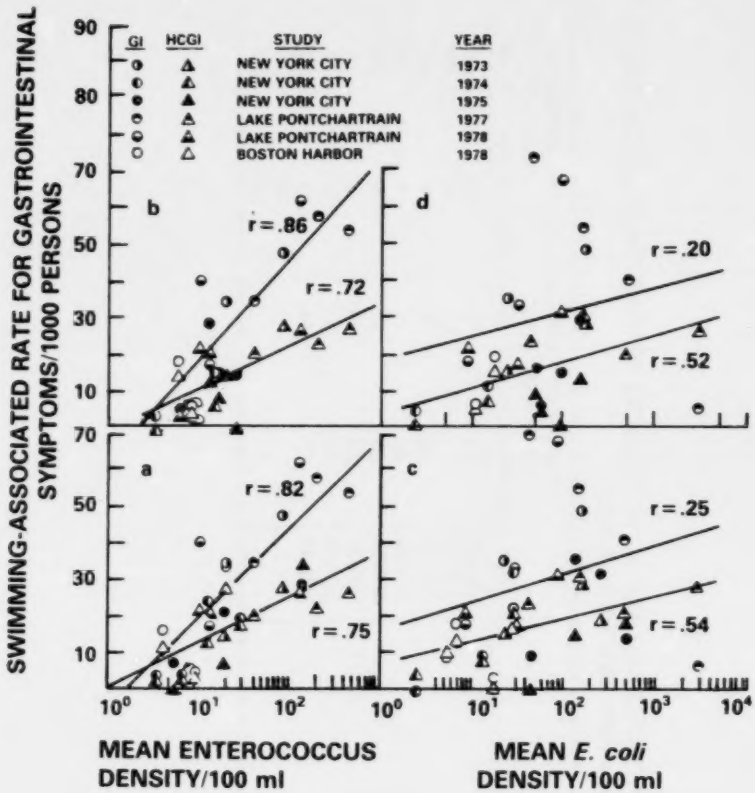

Figure 7. Swimming-associated rates for GI symptoms against the mean enterococcus and *E. coli* densities in the water. Data from all U.S. studies. Values for the points given in Tables 6-9. Definition of highly credible GI symptoms given in text, as is the rationale for clustering the trials. See Figure 2 for the meanings of a, b, c, and d. The actual trials clustered are shown in Tables A8 through A31, A44, A46, A47 and A50 in Appendix A.

are given in Tables 6-9 and the results of the regression analyses are given in Table 10. It is obvious that enterococcus densities in the bathing water provide the most meaningful and useful relationshp to the observed GI symptomatology. The formulae for the two pairs of enterococcus regression lines, the correlation coefficients (r) for the lines, and the corresponding p values are given in Table 10 along with the equations obtained by averaging the slopes and intercepts of each pair of lines. The "fits" for quadratic equations were no better than those for linear equations. These lines are shown in Figure 8 along with the 95 percent confidence limits around the lines. These were obtained from the data for the clustered trials. The confidence limits of the predicted rates for the clustered trials are given in Table A51.

The Y and X regression lines, given in Table 10 for enterococcus and shown with their confidence limits in Figure 8, predict the illness rates from the indicator densities. However, as noted earlier in this report, the conceptual framework for the program was that a decision would be made as to the *acceptable* risk level and this would be extrapolated to

TABLE 6. SUMMARY OF THE MEAN ENTEROCOCCUS DENSITY—GASTROINTESTINAL SYMPTOM RATE RELATIONSHIPS OBTAINED FROM CLUSTERED TRIALS FOR ALL THE U.S. STUDIES (INPUTS TO THE REGRESSION ANALYSIS, FIGURE 7a, TABLE 10)

Study	Beach	Year	Enterococcus Density per 100 ml	N		Symptom Rates in Cases Per 1000					
						Total Gastrointestinal			Highly Credible GI		
				Swim	Nonswim	Swim	Nonswim	▲[1]	Swim	Nonswim	▲
NYC[2]	Rock[5]	1973[8]	21.8	484	197	81	46	35	30.4	15.2	15.2
	C. Is.[6]		91.2	474	167	72	24	48*	46.4	18.0	28.4
		1974	3.6	1391	711	27	23	4	7.6	4.2	3.4
			7.0	951	1009	38	34	4	10.5	6.9	3.6
			13.5	625	419	42	17	25*	16.0	2.4	13.6
			31.5	831	440	43	23	20	18.1	—	18.1*
		1975	5.7	2232	935	63	55	8	18.8	19.3	-0.5
			20.3	1896	678	59	37	22*	14.8	7.4	7.4
Lake Pont.[3]	Levee	1977	154	579	191	60	31	29	34.5	—	34.5*
			44	874	451	86	51	35*	32.0	11.1	20.9*
			224	720	456	108	50	58**	31.9	8.8	23.1*
			495	895	464	108	54	54**	35.8	8.6	27.2**
	Levee	1978	11.1	1230	415	75	34	41**	36.6	14.5	22.1*
	Font.[7]		14.4	248	303	81	63	18	44.3	23.1	21.2
	Levee		142	801	322	112	50	62**	42.4	15.5	26.9*
Boston H.[4]	Revere	1978	4.3	697	529	83	66	17	23	11	12
	Nahant		7.3	1130	1099	71	67	4	33	28	5
	Revere		12.0	222	376	108	74	34*	41	13	28*

[1] Difference (swimmer rate minus nonswimmer rate).
[2] New York City, NY.
[3] Lake Pontchartrain, LA.
[4] Boston Harbor.
[5] Rockaways.
[6] Coney Island.
[7] Fontainebleau.
[8] Study population too small to cluster trials by similar indicator densities.
*p<0.05; **p<0.01

TABLE 7. SUMMARY OF THE MEAN ENTEROCOCCUS DENSITY—GASTROINTESTINAL SYMPTOM RATE RELATIONSHIPS OBTAINED FROM TRIALS GROUPED BY BEACH AND YEAR FOR ALL U.S. STUDIES (INPUTS TO THE REGRESSION ANALYSIS, FIGURE 7b).

Study	Beach	Year	Enterococcus Density per 100 ml	N		Symptom Rates in Cases Per 1000 Study					
						Total Gastrointestinal			Highly Credible GI		
				Swim	Nonswim	Swim	Nonswim	▲[1]	Swim	Nonswim	▲
NYC[2]	Rock[5]	1973	21.8	484	197	81	46	35	30.4	15.2	15.2
	C. Is.[6]		91.2	474	167	72	24	48*	46.4	18.0	28.4
	Rock.	1974	3.5	2767	2156	39	35	4	12.0	12.0	0.0
	C. Is.		16.4	1961	1185	42	26	16*	16.0	9.3	6.7
	C. Is.(1)	1975	17.9	1534	590	70	54	16	21.2	12.6	8.6
	(2)		27.7	1744	623	57	42	15	21.8	22.5	−0.7
	(3)		6.7	1131	475	50	44	6	13.7	8,5	5.2
	(4)		14.2	298	96	60	31	29	23.5	10.4	13.1
Lake Pont.[3]	Levee[8]	1977	44	874	451	86	51	35*	32.0	11.1	20.9*
			224	720	456	108	50	58**	31.9	8.8	23.1*
			495	895	464	108	54	54**	35.8	8.6	27.2**
	Levee[8]	1978	11.1	1230	415	75	34	41**	36.6	14.5	22.1*
			142	801	322	112	50	62**	42.4	15.5	26.9*
	Font.[7]		14.4	248	303	81	63	18	44.3	23.1	21.2
Boston H.[4]	Revere	1978	6.3	919	905	89	70	19	27.0	12.0	15.0
	Nahant		7.3	1130	1099	70	64	6	33.0	28.0	5.0

[1-7] See Table 6 for abbreviations.

[8] Data from Levee Beach were only clustered by trials with similar indicator densities for reasons explained in text.

*$p<0.05$; **$p<0.01$

TABLE 8. SUMMARY OF THE MEAN *E. coli* DENSITY—GASTROINTESTINAL SYMPTOM RATE RELATIONSHIP OBTAINED FROM CLUSTERED TRIALS FOR ALL THE U.S. STUDIES (INPUTS TO THE REGRESSION ANALYSIS FIGURE 7c)

Study	Beach	Year	*E. coli* Density per 100 ml	N		Symptom Rates in Cases Per 1000					
						Total Gastrointestinal			Highly Credible GI		
				Swim	Nonswim	Swim	Nonswim	▲[1]	Swim	Nonswim	▲
NYC[2]	Rock.[5]	1973[8]	24.8	484	197	81	46	35	30.4	15.2	15.2
	C. Is.[6]		174.0	474	167	72	24	48*	46.4	18.0	28.4
		1974	2.2	2514	1641	25	34	−9	8.0	3.7	4.3
			13.3	1304	1045	38	29	9	14.1	5.7	8.4*
			30.5	600	425	65	33	32*	23.3	2.4	20.9
		1975	46.8	1945	1099	55	51	4	13.4	17.8	−4.4
			142	775	194	76	41	35	24.5	10.3	14.2
			278	1049	330	55	24	31*	21.0	3.0	18.0*
			514	937	271	68	55	13	24.5	7.4	17.1
Lake Pont.[3]	Levee	1977	44	372	222	132	45	87**	32.3	9.0	23.3
			161	910	306	120	65	55**	52.7	22.8	29.9*
			497	574	307	85	45	40*	32.8	13.0	19.8
			3091	419	204	88	83	5	31.0	4.9	26.1
	Font.[7]	1978	9.0	248	303	81	63	18	44.3	23.1	21.2
	Levee		32.6	1123	382	78	44	34*	38.3	20.9	17.4
			93.7	918	355	103	36	67**	39.2	8.5	30.7
Boston H.[4]		1978	5.5	541	874	72	63	9	39	29	10
			7.0	477	410	86	68	18	23	10	13
			17.5	589	225	70	67	3	27	27	0
			29.5	442	495	93	71	22	32	14	18

[1-8] See Table 6 for abbreviations.

*p<0.05; **p<0.01

TABLE 9. SUMMARY OF THE MEAN *E. coli* DENSITY—GASTROINTESTINAL SYMPTOM RATE RELATIONSHIP OBTAINED FROM TRIALS GROUPED BY BEACH AND YEAR FOR ALL U.S. STUDIES (INPUTS TO THE REGRESSION ANALYSIS, FIGURE 7d)

Study	Beach	Year	*E. coli* Density per 100 ml	N		Symptom Rates in Cases Per 1000					
						Total Gastrointestinal			Highly Credible GI		
				Swim	Nonswim	Swim	Nonswim	▲[1]	Swim	Nonswim	▲
NYC[2]	Rock.[5]	1973	24.8	484	197	81	46	35	30.4	15.2	15.2
	C. Is.[6]		174.0	474	167	72	24	48*	46.4	18.0	28.4
	Rock.	1974	2.4	2767	2156	39	35	4	12.0	12.0	0
	C. Is.		15.3	1961	1185	42	26	16*	16.0	9.3	6.7
	C. Is.(1)	1975	52.4	1534	590	70	54	16	21.2	12.6	8.6
	(2)		98.6	1744	623	57	42	15	21.8	22.5	−0.7
	(3)		61.3	1131	475	50	44	6	13.7	8.5	5.2
	(4)		157	298	96	60	31	29	23.5	10.4	13.1
Lake Pont.[3]	Levee[8]	1977	44	372	222	132	45	87**	32.3	9.0	23.3
			161	910	306	120	65	55**	52.7	22.8	29.9*
			497	574	307	85	45	40*	32.9	13.0	19.8
			3091	419	204	88	83	5	31.0	4.9	26.1
	Font.[7]	1978	9.0	248	303	81	63	18	44.3	23.1	21.2
	Levee[8]		32.6	1123	382	78	44	34*	38.3	20.9	17.4
			93.7	918	355	103	36	67**	39.2	8.5	30.7*
Boston H.[4]	Revere	1978	18.0	919	905	89	70	19	27.0	12.0	15.0
	Nahant		11.5	1130	1099	70	64	6	33.0	28.0	5.0

[1-8] See Table 7 for abbreviations and notations.

*$p<0.05$; **$p<0.01$

a mean indicator density limit to be used as a guideline. This requires the regression of X on Y. These lines along with their confidence limits, correlation coefficients and formulae are given in Figure 9. The 95 percent confidence limits for the mean enterococcus densities predicted for the observed swimming-associated rates are given in Table A52. The author favors the use of the criteria for HCGI symptoms because of the greater credibility of its data base and because it is more conducive to economic analysis. The 95 percent confidence limits for the regression lines as shown (Figure 9) are rather broad although the slopes are significantly different from zero. This was not unexpected since the relationshps obtained are generalizatons which may be altered by any of a number of temporal and spacial factors relative to the indicator, the pathogen, the relationship of the pollution sources to the bathing beach, the levels of the specific illnesses in the overall population, and the immune status of the swimmers. These will be discussed in the next two sections.

Examination of the illness-indicator relationships by location and by year at a given location could provide some insight as to possible spatial and temporal effects. The latter was not attempted because of the small number of points available for analysis by year. The regression lines for the New York City study were compared to those obtained from

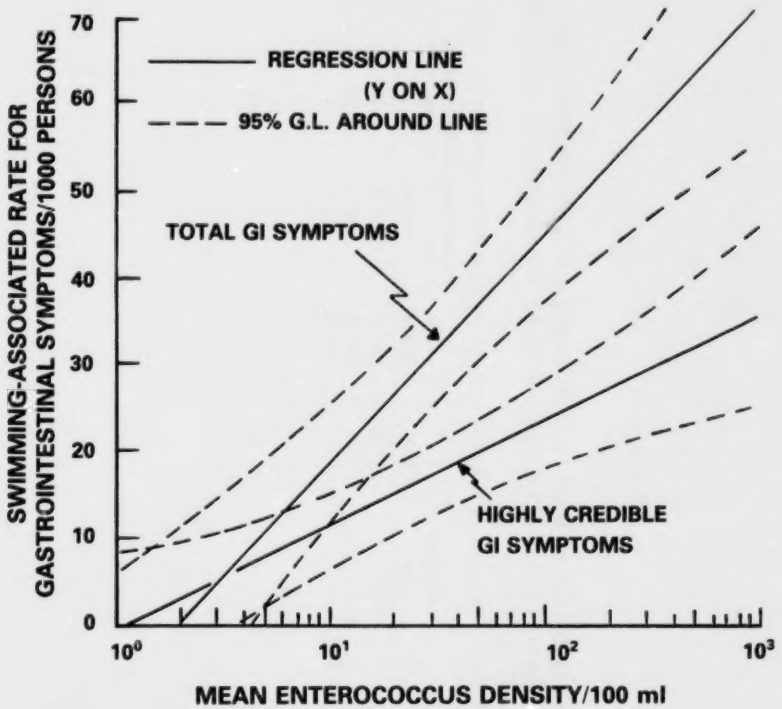

Figure 8. Regression lines for swimming-associated GI symptom rates (Y) against the mean enterococcus densities in water (X). Lines drawn from averages of slopes and intercepts from Figures 7a and 7b. Confidence limits are those for the regression lines shown in Figure 7a. Representation predicts the illness rates from the indicator densities and presents the 95% confidence limits of the former.

TABLE 10. REGRESSION FORMULAE AND CORRELATION COEFFICIENTS FOR SWIMMING-ASSOCIATED GI SYMPTOMS AGAINST ENTEROCOCCUS DENSITIES AND AGAINST *E. coli* DENSITIES IN THE BATHING WATERS (ALL U.S. STUDIES)

Indicator	Analysis by	N	Gastrointestinal Symptoms				HCGI Symptoms[1]			
			Slope	Intercept	r	p	Slope	Intercept	r	p
Enterococcus	Trials	18	24.19	−5.09	.82	<.001	12.17	0.20	.75	<.001
	Summers	16	27.37	−9.52	.86	<.001	11.53	−1.36	.72	<.005
Average			25.78	−7.31			11.85	−0.58		
E. coli	Trials	20	7.37	15.73	.25		6.30	5.88	.54	
	Summers	17	6.63	17.72	.20		7.30	2.79	.52	

[1] Highly credible gastrointestinal symptoms.

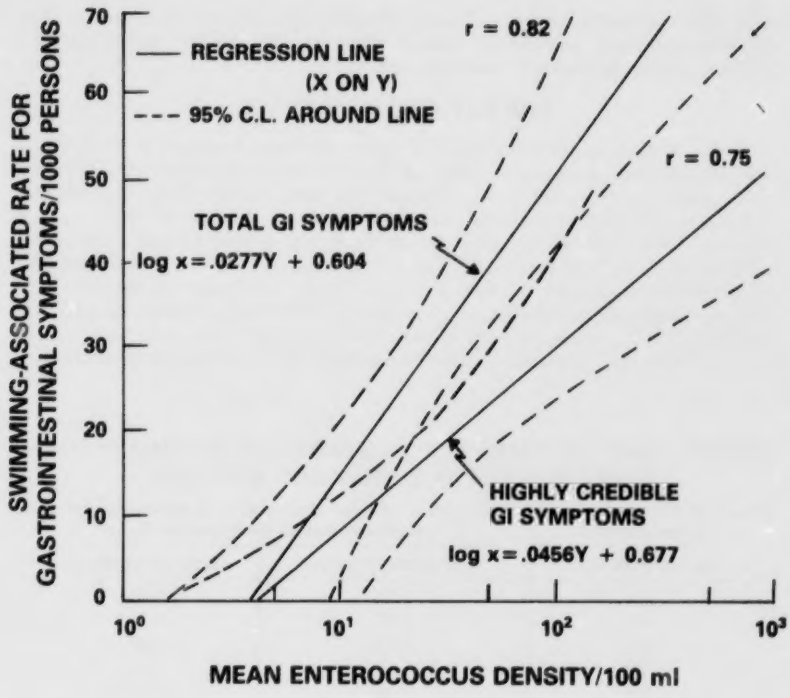

Figure 9. Health effects criteria for marine recreational waters developed by the USEPA epidemiological-microbiological program. Criteria are X on Y regression lines of the mean enterococcus density in the water against the swimming-associated rate of gastrointestinal symptoms. Lines drawn in the same manner as those shown in Figure 8. The 95% confidence limits around the lines are those for data given in Table 6.

the combination of the Lake Pontchartrain and the Boston Harbor studies; however, even for the trials clustered by similar indicator densities (Table 6), each line was defined by only nine points. Significant differences were obtained between the lines for highly credible but not total GI symptoms. The lines for total GI symptoms were not significantly different; however, those for HCGI symptoms were, although the two lines stay virtually within the 95 percent confidence limits of the total data. This provides some basis for the generalization obtained from the single regression line. This generalization may not be totally accurate in all situations. Thus, in the present case, the sources of pollution to the beaches in the Lake Pontchartrain and Boston Harbor studies were ill-defined and, presumably, more distant. This and the effect of the immune status of the swimming population could explain the significant differences between the regression lines for highly credible but not total GI symptoms. In any event, these results emphasize the conclusion that guidelines derived from these criteria cannot be used without judgment; rather, they must be used in concert with good public health practice (e.g., taking into consideration changes in the incidence of enteric disease in the discharging population), an environmental (sanitary) survey, and judgment with regard to their limitations in time and space. In fact, the correlations obtained are remarkably

good when the sources of temporal and geographic variability are considered, and this has some interesting implications concerning the agent(s) and host population, i.e., ubiquity, infectivity, survival, immunity, etc.

THE ETIOLOGIC AGENT(S)

When the study design for the EPA program was being developed in 1969-1970, it was thought that swimming in sewage-polluted waters would constitute a relatively minor route of transmission for GI illness and that relatively high levels of pollution (as indexed by microbial indicator densities) would be required before GI illness could be detected. These assumptions were made on the basis of existing notions and available information (8,24). Both these assumptions were incorrect. If the nonswimming rates for GI symptomatology can be considered as those for the population at large, then swimming in sewage-polluted waters constitutes a significant route of transmission for the illnesses obtained, at least for individuals of "swimming age." This can be seen from the tabular (Table 11) and graphic (Figure 10) representations of the ratios of the rates for

TABLE 11. RATIO OF SWIMMER TO NONSWIMMER GASTROINTESTINAL SYMPTOM RATES BY ENTEROCOCCUS DENSITY[1]

Enterococcus	Swim/Nonswim GI Symptom Rate	
Density/100 ml	Total Gastrointestinal	Highly Credible GI
3.6	1.17	1.81
4.3	1.26	2.09
5.7	1.15	0.97
7.0	1.12	1.52
7.3	1.06	1.18
11.1	2.21	2.52
12.0	1.46	3.15
13.5	2.47	6.67[2]
14.4	1.29	1.92
20.3	1.59	2.00
21.8	1.76	2.00
31.5	1.87	I[3]
44.0	1.69	2.89
91.2	3.00	2.58
142.0	2.24	2.74
154.0	1.94	I[3]
224.0	2.16	3.63
495.0	2.00	4.13

[1] Data taken from Table 6.
[2] Due to unusually low nonswimmer rate.
[3] Indeterminate because of no cases among nonswimmers.

swimmers divided by those for nonswimmers against the enterococcus densities for the clustered trials. In fact, at enterococcus densities of 70 and 10/100 ml, respectively, the rates for total and HCGI symptoms among swimmers were twice those for nonswimmers, and they are projected to be equal (a ratio of "1") at an enterococcus density of about 1/100 ml. This suggests that the etiologic agent(s) for the observed GI symptomatology is present in sewage in large numbers, that it is highly infective and/or that it survives sewage treatment, disinfection and/or transport better than the indicator.

One of the desired outputs from the program was an answer to the question: Does the swimming-associated illness rate increase with the levels of these specific illnesses in the population at large? This relationship was not observed for the types of illnesses obtained in this study (Table 12), probably because of the high level of immunity to the agent in the population.

Initially, it was thought that the Egyptian data could be used in the derivation of the final criteria. By the end of the first year of the Egyptian study, it was obvious that the data from the Alexandria residents could not be so used, and by the end of the third year, it was concluded that this was also true of data from the Cairo visitors. The regression lines for the rates of swimming-associated vomiting and diarrhea from these two groups along with those for GI and HCGI symptoms from the United States studies against the corresponding mean enterococcus densities are presented in Figure 11. It can be seen that, in the United States studies, gastrointestinal illness rates comparable to those obtained in the Egyptian study were associated with bathing in waters with much lower enterococcus densities. Part of the dissimilarity is probably due to differences in the nature (raw vs. treated) and proximity of the pollution sources in the United States and Egyptian studies. However, disparities in the immune state of the populations to the etiologic agent(s) probably accounts for most of the differences in the indicator-illness relationships obtained.

The importance of immunity in the epidemiology of the swimming-associated gastroenteritis is also supported by the age distribution of the attack rates. In most of the studies, children (<10 years of age) were found to have the highest symptom rates.

The following characteristics of the swimming-associated illness were obtained in or can be inferred from the findings of the EPA program: (i) The illness is a relatively benign gastroenteritis with a short incubation period (Figure 12), acute onset, short dura-

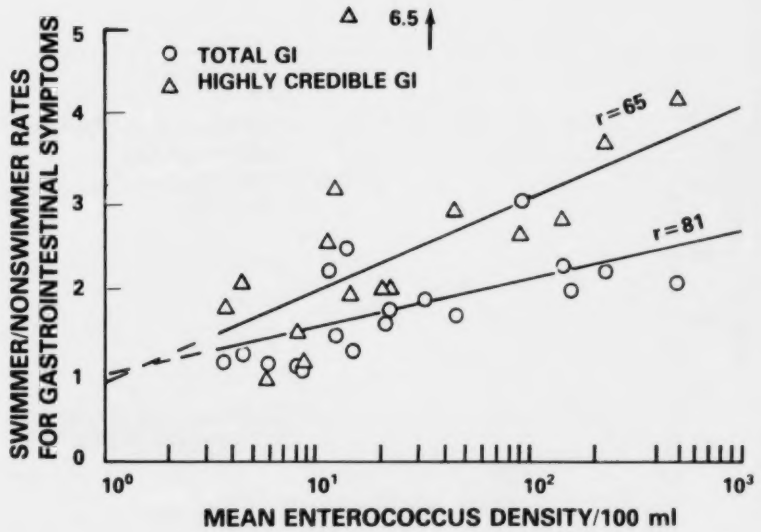

Figure 10. Ratios of swimmer to nonswimmer rates of gastrointestinal symptoms against the mean enterococcus density in the water. Data from Table 6. One value not used in the calculations.

TABLE 12. RELATIONSHIP OF SWIMMING-ASSOCIATED (▲) TO BACKGROUND (NONSWIM) RATES FOR GASTROINTESTINAL SYMPTOMS

Enterococcus Density[1]	Rates Per 1000 Persons			
	Total GI		Highly Credible GI	
	Nonswim	▲	Nonswim	▲
3.6-7.0	23	4	4.2	3.4
	34	4	6.9	3.6
	55	8	19.3	−0.5
	66	17	23.0	11.0
	67	4	28.0	5.0
11.1-21.8	17	25	2.4	13.6
	34	41	13.0	28.0
	37	22	14.5	22.1
	46	35	14.8	7.4
	63	18	15.2	15.2
	74	34	23.1	22.1
91.5-154	24	48	15.5	26.9
	31	29	18.0	28.0
	50	62	—[2]	34.5

[1] Values ordered according to the nonswimming rate within a density cluster. Only clusters of 3 or more reasonably close values used.
[2] Nonswimming rate "0."

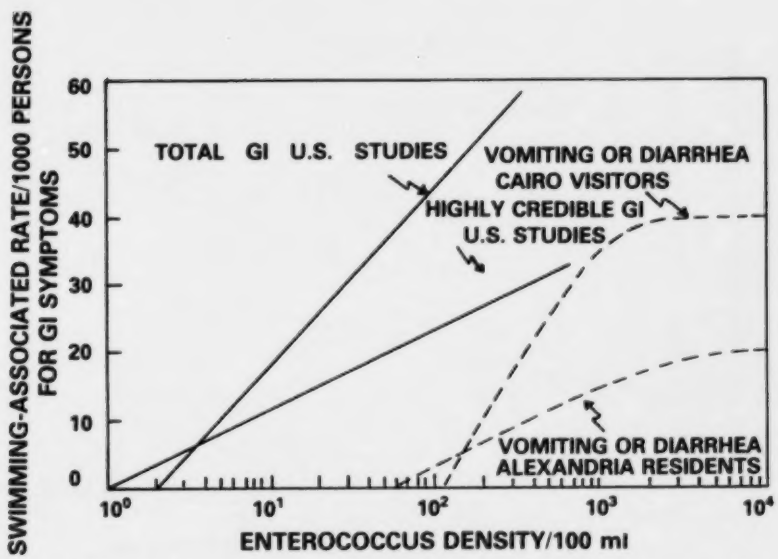

Figure 11. Comparison of the illness-indicator relationship obtained from the U.S. studies with those for the Cairo visitors and Alexandria residents in the Egyptian studies. Those for the U.S. populations taken from Figure 7 and those for the Egyptian study from Figure 4.

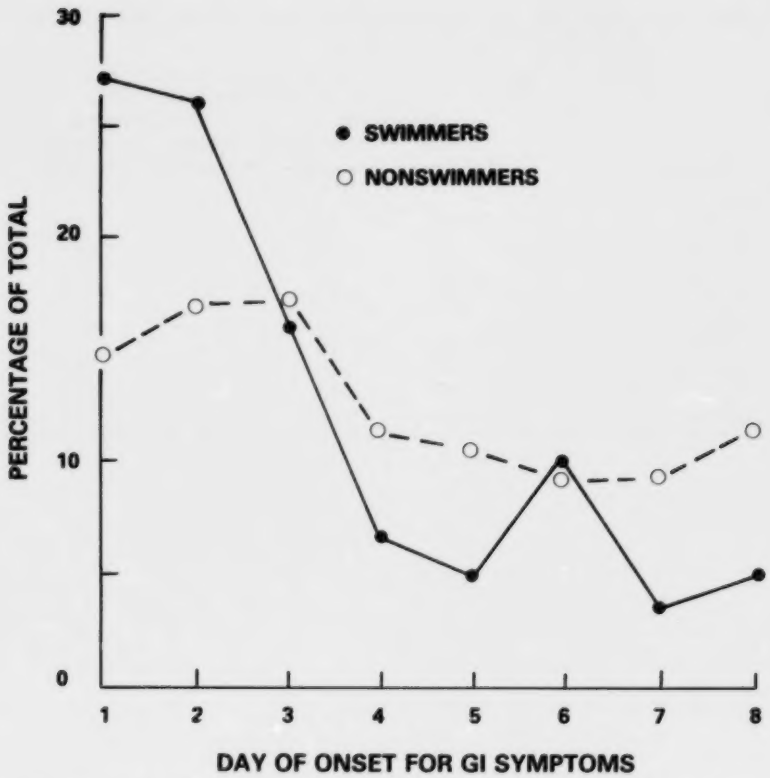

Figure 12. Day of onset of GI symptoms as obtained from the 1975 New York City trials.

tion (Table 13) and rare, if any, sequelae. (ii) It is widely distributed; most individuals are immune, and, in general, children have the highest attack rates. (iii) The etiologic agent is highly infectious, is present in sewage in large numbers, and/or survives sewage treatment disinfection and transport in the marine environment somewhat better than the indicators. These considerations suggest the human rotavirus and/or the parvo-like viruses as the etiologic agents.

There are at least three explanations for the observations that individuals who swim during several days in a given week (from the Egyptian study) or for prolonged periods during a given day (from the New York City study) have low GI symptom rates. The obvious one is that these are "healthier" individuals. The second assumes that the extent of swimming is correlated with age, that is, individuals who swim regularly and extensively are more experienced and ingest less of the bathing water. However, it is commonly assumed that children are in the water the longest and also ingest the most water. The third explanation requires that the illnesses involved have short (< 3 days) incubation periods and that there be a good immunity to the etiological agents. The rationale for the Egyptian observations is that the susceptible individuals become ill within a day or so of the time they start swimming.

TABLE 13. DURATION OF GASTROINTESTINAL SYMPTOMATOLOGY: NEW YORK CITY, 1975 TRIALS

Symptom	Duration of Symptoms in Days for Swimmers		Nonswimmers	
	Number Reporting	Average Duration	Number Reporting	Average Duration
Total				
Vomiting	30	2.8	10	2.6
Diarrhea	73	2.6	26	2.7
Stomachache	101	2.7	36	2.4
Nausea	64	2.7	18	2.8
Disabling				
Vomiting	17	3.7	5	2.6
Diarrhea	22	3.0	11	3.2
Stomachache	36	3.5	12	3.0
Nausea	24	2.6	8	3.2

SECTION 7
LIMITATIONS IN THE USE OF THE RECOMMENDED CRITERIA

The criteria presented in this report (the enterococcus density in the bathing water against the swimming-associated rates for total and HCGI symptoms) are generalizations which have been found to apply in a number of situations. Nevertheless, a number of considerations, including the limitations in the indicator concept itself, impact on the use of the criteria as well as the guidelines and standards derived therefrom. More important, these considerations require that the findings from monitoring programs be interpreted in the light of good public health and environmental practice. They have been described elsewhere (49,66) and several of the more important ones will be considered herein.

SMALL POINT SOURCES

The rationale for the use of guidelines and standards based on fecal indicator densities for indexing the health hazards in sewage polluted waters is that, under average conditions of illness in the discharging population, there is a reasonably constant indicator to pathogen ratio in the sewage and its receiving waters. Thereby, an acceptable probability of illness caused by the pathogen can be extrapolated to a given indicator density, which is then recommended as a guideline and promulgated as a standard. Such relationships appear to hold for waters receiving the discharges from relatively large municipal sewage treatment facilities. However, as the number of individuals who contribute to the source of the fecal wastes becomes smaller and smaller, the indicator-pathogen ratio will vary more and more from the average upon which the guideline or standard is based. In the extreme case where the fecal wastes of a single ill individual or carrier are discharged into the water, the number of pathogens may equal or exceed the number of indicator microorganisms. Routine examination of such waters for fecal indicators would be of no value. Furthermore, the routine examination for the pathogens would not be especially useful since the release of enteric pathogens will be sporadic. The solution is administrative action prohibiting such discharges into recreational waters.

ILLNESS RATES IN THE DISCHARGING POPULATION

Most epidemiologists and health officers recognize that, under epidemic conditions, the actual indicator-pathogen ratio may change sufficiently from that upon which a guideline was based so that the acceptable risk of illness will be exceeded unless the guideline is temporarily made more restrictive. The recent swimming-associated outbreak of shigellosis on the Mississippi River below Dubuque, Iowa (15) appears to represent an instance where, although the 200/100 ml fecal coliform guideline was probably exceeded, the outbreak did not occur until there was a large enough number of ill individuals and carriers in the discharging population.

Conversely, if there is a significant and consistent decrease in the illness rate in the discharging population over a prolonged period of time, the rate for that specific illness associated with an existing indicator guideline or standard may be considerably less than predicted. The absence of recreational water-associated salmonellosis probably represents a case in point.

FECAL INDICATORS VERSUS PATHOGENS

The use of fecal indicators such as coliforms or portions of the coliform population, fecal streptococci, and *C. perfringens* for indexing the health hazards in drinking and recreational waters dates back to the late 1800s and early 1900s (32). This occurred shortly after these organisms were first isolated and associated with the fecal wastes of warm-blooded animals. Within the context of the limitations being discussed, such practices were and are sound both on theoretical and practical grounds since it is recognized that (i) there are a large number of pathogenic bacteria and viruses potentially present in municipal sewage (67,68), each with its own probability of illness associated with a given dose; (ii) monitoring for each of the pathogens on a routine basis would be a herculean task; (iii) enumeration methods for some of the more important pathogens are unavailable and for the rest are difficult; (iv) pathogen density data are difficult to interpret because the methodology generally is imprecise and inaccurate and because of the meager dose-response data available; and (v) on theoretical grounds, the intent is not to index the presence of the pathogen but rather its potential to be there in sufficient numbers to cause unacceptable health effects.

By no means should the foregoing be construed as suggesting that recreational water quality criteria and the derived guidelines are unnecessary. To the contrary, criteria amenable to risk analysis are absolutely essential. It is evident from the nature of the illness indicator (Y on X) lines and the heavy usage of estuarine and coastal beaches in the United States that large numbers of individuals are becoming ill as a consequence of swimming in sewage-polluted waters. Furthermore, as seen from the Dubuque outbreak (15), the potential for more serious illness exists. Nevertheless, since the illnesses involved are relatively benign, there is undoubtedly a rate which is acceptable; however, *the acceptances of the risks involved should be deliberate decisions with consideration of all the factors involved and with local input.*

A temporary consequence of the application of the criteria may be the withdrawal of certain recreational resources from public use. However, the long range impact should be pollution abatement. This requires better technology for obtaining the data base needed for the translation of the target area criteria which have been developed into effluent guidelines on a case-by-case basis.

The findings from the EPA program have raised a number of questions. One is the nature of the etiologic agent for the gastrointestinal symptomatology. A second is the need for a more human fecal specific and environmentally resistant indicator. This relates to the difficult question of stormwater runoff and nonpoint sources. The third is need for separate criteria for fresh waters. Studies in progress which address these questions should be continued.

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APPENDIX

TABLE A1. TOTAL AND FECAL COLIFORM STANDARDS FOR PRIMARY CONTACT RECREATIONAL WATERS AS OF 1978

State[a]	Year Rev[b]	Water Type[c]	Total Coliform Limit per 100 ml			Fecal Coliform Limit per 100 ml		
			Average	Percentile	One Sample	Average	Percentile	One Sample
Alabama[1,2,3]	77	SW				LM[d] 100		
	77	SW				LM 200		
Alaska	79	ALL				Mean 20	90%≤40	
Arizona	73	FW				EPA	EPA	
Arkansas	77	FW				EPA	EPA	
California	78	SW	Ave 1000	80≤1000		EPA	EPA	
	76	FW	Med[e] 240		≤10,000	Med 50	90%≤400	
Colorado	75	FW				EPA	EPA	
Connecticut[4,5,6]	76	SW	Med 700	90%≤2300		EPA[f]	90%≤500[g]	
	76	FW	Med 1000	80%≤2400		EPA[f]	96%≤500[g]	
Delaware	75	ALL				EPA		
District of Columbia	Pro[h]	ALL				EPA	EPA	
Florida	74	ALL	LM 1000	80≤1000	≤2400	EPA	EPA	≤800
Georgia[1,2]	77	SW				LM 100[2e]		
	77	FW				LM 200		
Hawaii	74	ALL	Med 1000	90%≤2400		EPA	EPA	
Idaho	Pro	FW				LM 50	90%≤200	≤500
Illinois	75	FW				EPA	EPA	
Indiana[7]	78	FW				EPA		≤400[a,i]
Iowa[8]	77	FW				EPA	EPA	
Kansas	78	FW				EPA	EPA	
Kentucky[9,10]	76	FW	Ave 1000	80%≤1000	≤2400	EPA[11]	EPA[11]	
Louisiana	77	ALL				EPA	EPA	

Table A1. (continued)

State[a]	Year Rev[b]	Water Type[c]	Total Coliform Limit per 100 ml			Fecal Coliform Limit per 100 ml		
			Average	Percentile	One Sample	Average	Percentile	One Sample
Maine	77	SW	Med 70	90%≤230		Med. 1000	90%≤200	
	77	FW				NTE 200[g,j]		
Maryland[12]	74	ALL				EPA	EPA	
Massachusetts[13]	78	SW	Med 700	90%≤1000				
	78	FW				EPA	EPA	
Michigan[27]	73	FW				EPA		
Minnesota[14]	77	FW				EPA	EPA	
Mississippi	77	ALL				EPA	EPA	
Missouri[8,15]	77	FW				EPA	EPA	
Montana	78	FW				EPA	EPA	
Nebraska	77	FW				EPA	EPA	
Nevada	74	FW				EPA	EPA	
New Hampshire[9]	77	ALL			240			
New Jersey[6]	74	ALL				EPA		
New Mexico[16]	77	FW				LM 100	90%≤200	
New York[9,17,18]	74	ALL	Med 2400	80%≤5000		EPA		
North Carolina[19,20]	77	ALL				EPA[16]	80%≤400	
North Dakota	77	FW				EPA	EPA	
Ohio[21]	78	FW				EPA	EPA	
Oklahoma	76	FW				EPA	EPA	
Oregon[22]	Pro	SW	Ave 240	80%≤240				
		FW	Ave 1000	80%≤2400				
Pennsylvania[18]	Pro	ALL				EPA		
Rhode Island	77	SW	Med 700	90%≤2300		Med 50		90%≤500
	77	FW	Med 100	80%≤2400		Med 200[g]		80%≤500[g]
South Carolina	77	ALL				EPA	EPA	
South Dakota	78	FW				EPA	80%≤200	≤400

Table A1. (continued)

State[a]	Year Rev[b]	Water Type[c]	Total Coliform Limit per 100 ml Average	Percentile	One Sample	Fecal Coliform Limit per 100 ml Average	Percentile	One Sample
Tennessee[1,23,24]	77	FW				EPA		≤1000
Texas[6]	76	ALL				EPA	EPA	
Utah	78	FW	LM 1000			EPA		
Vermont	78	FW	NTE 500			NTE 200		
Virginia	77	ALL				EPA	EPA	
Washington	77	SW				MED 14	90%≤43[25]	
	77	FW				LM 100	90%≤200[16]	
West Virginia	77	FW	Ave 1000	80%≤1000	≤2400	EPA	EPA	
Wisconsin[28]	78	FW				EPA	EPA	
Wyoming	78	FW				EPA	EPA	
Puerto Rico	76	ALL				EPA	80%≤400	
Virgin Islands	73	ALL				LM 70		
Trust Territory	73	ALL				EPA	EPA	
American Samoa	73	ALL				Ave 100	90%≤200	
Guam[9]	76	ALL				Ave 200	EPA	

[a] Does not include all the caveats, special requirements, limitations, etc.
[b] Year of latest revision.
[c] SW - seawater (estuarine and coastal); FW - freshwater.
[d] Log mean.
[e] Median.
[f] Geometric mean not to exceed 200/100 ml.
[g] Guideline.
[h] Proposed.
[i] In one month.
[j] Not to exceed.

[1] Waters in vicinity of STP outfall not suitable.
[2] Designated as "coastal" and "all other recreational waters."
[3] If standard exceeded, waters considered acceptable if a second sanitary survey and evaluation indicates no significant public health risk.
[4] For listed rivers, disinfection of STP effluents required; and standards only apply between months of May through September.
[5] "Coliform bacteria . . . are related to the probability of contamination by undisinfected sewage. High results may be due to soil bacteria or bacteria from the feces of warm-blooded animals which are not of sanitary significance."
[6] Sanitary surveys required.
[7] Applies only from April through October.
[8] Applies April 1 - October 31.
[9] Unless naturally occurring.
[10] If TC exceeded, then FC is used.
[11] Only applicable from May through October.
[12] Waters exceeding standard acceptable only if sanitary survey shows no significant public health risk.
[13] Except as provided in Regulation 2.1.
[14] Standards relate only to intrastate waters.
[15] Except when affected by stormwater runoff.
[16] Varies with body of water; standard as given used in most cases, EPA guideline used in a few.
[17] Applies only when disinfection is practiced.
[18] For "International Boundary Waters" under Great Lakes Water Quality agreement of 1972, log mean TC 1000/100 ml and FC 200/100 ml.
[19] Applicable only during May through September.
[20] Not applicable during or immediately following periods of rainfall.
[21] Where there are no lifeguards and/or bathhouse facilities, log mean of 1000/100 ml and 90% ≤2000/100 ml apply.
[22] ...acterial pollution or other conditions deleterious to waters used for . . . bathing . . . or otherwise injurious to public health shall not be allowed."
[23] <1/100 ml set as 1/100 ml in calculating log mean.
[24] Individual samples cannot be collected within 12 hours of each other.
[25] Standard given is for Class A (Excellent) waters which ". . . shall meet or exceed the standards for all or substantially all uses . . ." Class AA (Extraordinary) fresh water standard is a median TC of 50/100, 90% ≤100/100 ml. Class B (Good) for fresh water is median FC of 200/100 ml, 90% ≤400/100 ml; for sea waters, the standard is the same as that for Class A fresh waters.
[26] If water quality and sanitary surveys show 200/100 ml exceeded occasionally due to "natural causes," log mean of 300/100 ml in lakes and reservoirs and 500/100 ml in free flowing FW streams becomes the limit.
[27] Limits may be exceeded if due to "uncontrollable non-point sources."
[28] Sanitary survey to assure protection is chief criterion; bacterial limits are guidelines.

TABLE A2. DEMOGRAPHIC CHARACTERISTICS OF THE FOUR SUBPOPULATIONS FOR 1974 NEW YORK CITY TRIALS

Demographic Group	Percent of Respondents by Category			
	BA Beach		RU Beach	
	Swim (N = 1961)	Nonswim (N = 1185)	Swim (N = 2767)	Nonswim (N = 4156)
Sex				
Male	44.0	33.5	46.9	27.1
Female	56.0	66.5	53.1	62.9
Age Group				
0-9	24.9	10.1	26.7	26.4
10-19	36.1	12.7	21.3	11.7
20-39	14.4	65.2	43.1	47.7
≥40	24.6	12.0	8.9	14.4
Ethnic Group				
Hispanic-American	47.8	33.5	52.6	53.1
White	36.8	37.4	30.1	29.6
Black	15.4	29.1	17.3	17.3
Persons/rooms ratio[1]				
≤0.9	26.2	21.8	21.3	29.5
1.0-1.3	32.7	40.8	40.6	39.2
≥1.4	41.1	37.4	38.1	31.3

[1]Number of persons in household divided by number of rooms in household, as an indicator of socioeconomic status (SES), 0.9 or less persons/rooms indicates higher SES; 1.0-1.3, middle SES; and 1.4 or more, lower SES.
BA — barely acceptable; RU — relatively unpolluted.

TABLE A3. MEAN INDICATOR DENSITIES AT THE CONEY ISLAND AND ROCKAWAY BEACHES DURING 1973 AND 1974 TRIALS

Indicator	Log_{10} Mean Recovery/100 ml			
	1973		1974	
	Coney Island	Rockaway	Coney Island	Rockaway
Total coliforms	983*	39.8	1213*	43.2
Fecal coliforms	165*	21.5	565*	28.4
Escherichia coli	174*	24.8	15.3*	2.4
Klebsiella	122*	13.7	59.2*	3.5
Enterbacter-Citrobacter	530*	11.1	434	6.6
Fecal Streptococci	91.2	21.8	16.4*	3.5
Pseudomonas aeruginosa	30.4	6.5	45.8*	3.1
Aeromonas hydrophila	25.3	26.5	9.6	4.9
Vibrio parahaemolyticus	ND	ND	54.5	32.8

*Significantly different from density at Rockaways at 95 percent confidence level.

TABLE A4. SWIMMING ASSOCIATED SYMPTOM RATES FOR NEW YORK CITY BEACHES IN 1973, 1974

Symptom	Swimming Assoc. (swim - nonswim) Sympt. Rates[1] Rockaway, NYC 1973 (484-197)[2]	Rockaway, NYC 1974 (2767-2156)[2]	Coney Island, NYC 1973 (474-167)[2]	Coney Island, NYC 1974 (1961-1185)[2]
Vomiting	0	0	21*	4
Nausea	15	−2	26*	−1
Diarrhea	18	0	28*	8**
Stomachache	41**	0	39**	9**
Sore throat	47**	−3	18	−2
Bad cough	20	1	6	5
Chest cold	−3	−1	−2	−2
Nose	21	5	8	3
Ear	−3	6	−1	6*
Eye	28	3	24	3
Skin (exclusive of sunburn)	64**	7	113**	9**
Fever (100°F)	15	6*	6	4
Headache	6	−6	10	2
Backache	−8	−6	2	−1
Home due to symptom	−10	4	6	9*
In bed due to symptom	−6	1	−3	4
Medical help due to symptom	0	2	5	3

[1] Rates in cases per 1000 persons.
[2] ( ) = swim, nonswim.
*p< .1; **p< .05.

TABLE A5. SWIMMING ASSOCIATED RATES FOR SYMPTOM GROUPS AT THE NEW YORK CITY BEACHES (1973-74)

Symptom Groups[1]	Swimming Associated Rate (Per 1000 Persons) Rockaway, NYC 1973	Rockaway, NYC 1974	Coney Island, NYC 1973	Coney Island, NYC 1974
Gastrointestinal	35	5	48*	16*
Highly Credible GI	15	0.0	28	6.7
Respiratory	63*	4	27	8
"Other"	5	9	33	6
Disabling	4	4	17	9
Skin	64[2]	7	113[2]	9*

[1] See text for symptoms included in each group.
[2] Partly due to jellyfish stings.
*p≤.05; **p≤.01 for differences between swimmer and nonswimmer rates.

TABLE A6. COMPARISON OF *Salmonella* AND TOTAL COLIFORM DENSITIES (PER 100 ML) AT CONEY ISLAND AND ROCKAWAY BEACHES (ARRANGED IN DESCENDING ORDER OF COLIFORM VALUES)

Coney Island			Rockaway		
Date	Total Coliforms[1]	*Salmonella*[2]	Date	Total Coliforms[1]	*Salmonella*[2]
11 Aug.	14500	0.020	18 Aug.	350	<0.018
12 Aug.	3300	0.045	22 July	205	<0.018
19 Aug.	1850[3]	0.020	29 July	185	0.040
18 Aug.	1550	0.020	19 Aug.	90	<0.018
22 July	900	0.040	12 Aug.	70[3]	0.020
29 July	435	0.020	14 July	30	<0.018
28 July	360	0.020			
14 July	145	0.020			

[1] mC estimate of total coliforms from low-tide samples collected concurrently with those for the *Salmonella* assays.
[2] Obtained from examination of 55.5 liters by S-HVS method (46).
[3] Estimate obtained by MPN method.

TABLE A7. ANALYSIS OF GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GASTROINTESTINAL (HCGI) SYMPTOM RATES BY DEMOGRAPHIC GROUPING

Demographic Group	GI Symptom Rates Per 100 Persons							
	Barely Acceptable Beach				Relatively Unpolluted Beach			
	GI		HCGI[8]		GI		HCGI	
	Swim	Nonswim	Swim	Nonswim	Swim	Nonswim	Swim	Nonswim
Total sample	42[6]	26	16	9.3	39	35	12	12
Children[1]	57[6,7]	14	24[6]	<4.5	23[6]	55	9.2[6]	28
Hispanic-American	45[6,7]	17	21[6,7]	7.6	24	12	5.6	3.0
High-Middle persons/rooms[2] Ratio	42[6]	16	14[6]	5.2	41	34	15	10
Adults[3]	37	29	13	11	42	32	12	9.5
Non-Hispanic-Americans[4]	38	35	10	11	43	39	13	13
Lowest persons/rooms Ratio[5]	42	45	21	17	37	35	9.1	13

[1] ≤10 yrs. old;
[2] ≥1.0 persons/rooms ratio;
[3] >10 yrs. old;
[4] white and black;
[5] <1.0 persons/rooms ratio;
[6] significantly different (P-0.05) than nonswimming control;
[7] significantly higher (P-.05) than RU swimmers;
[8] All instances of vomiting, diarrhea with fever or a "disabling" response, and nausea and stomachache with fever.

TABLE A8. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO ENTEROCOCCUS DENSITIES

Year	Beach and Date[1]	Enterococcus Density/100 ml Mean	Range
1973[2]	R7/14, R7/22, R7/28, R7/29, R8/11 R8/12, R8/18, R8/19	21.8	1.2-59
	C7/14, C7/22, C7/28, C7/29, C8/11 C8/12, C8/18, C8/19	91.2	6-186
1974	R7/28, R8/18, R8/31	3.6	2-5
	R7/20, R7/21, C8/31	7.0	7
	C7/20, C7/28	13.5	10-17
	C7/21, C8/18	31.5	30-33
1975	A7/6, A7/5, A7/11, A7/19, A7/20 A7/27, B7/6, B7/19, B7/26, C6/19 C7/20, C7/27, D7/19, D7/20	5.7	2-11
	A8/2, B7/5, B7/27, B8/2, C7/5 C7/6, C7/26, C8/2, D8/2, D8/3	20.3	14-38
	A8/3, B7/20, B8/3, C8/3	154	86-298

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island; A — 18th-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island; C — 2nd-4th Streets, Brighton, Coney Island.
[2] 1973 trials clustered by beach.

TABLE A9. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO *E. coli* DENSITIES

Year	Beach and Date[1]	*E. coli* Density/100 ml Mean	Range
1973	R7/14, R7/22, R7/28, R7/29, R8/11 R8/12, R8/18, R8/19	24.8	3-34
	C7/14, C7/22, C7/28, C7/29, C8/11 C8/12, C8/18, C8/19	174	50-708
1974	R6/22, R7/21, R7/28, R8/18, R8/31 C8/31	2.2	1-4
	R7/20, C7/21, C7/28, C8/18	13.3	9-19
	C6/22, C7/20	30.5[2]	26-35
1976	A7/5, A7/6, A7/26, A7/27, B7/5, B7/6 B7/26, C7/5, C7/6, C7/26	46.8	22-89
	A7/19, A7/20, A8/3, C7/19, C7/20, D8/2	142	115-169
	A8/2, B7/20, B7/27, C8/2, C8/3 D7/19, D7/20, D8/3	278	208-356
	B7/19, B8/2, B8/3, C7/27	514	441-659

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island; A — 18th-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island; C — 2nd-4th Streets, Brighton, Coney Island.
[2] Arithmetic mean.

TABLE A10. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO FECAL COLIFORM DENSITIES

Year	Beach and Date[1]	Fecal Coliform Density/100 ml Mean	Range
1973	R7/14, R7/22, R7/28, R7/29, R8/11 R8/12, R8/18, R8/19	21.5	6.2-34
	C7/14, C7/22, C7/28, C7/29, C8/11 C8/12, C8/18, C8/19	165	49-431
1974	R7/28, R8/18	18[2]	17-19
	R6/22, R7/20, R7/21	38	29-50
	C7/20, C7/28	252[2]	231-273
	C7/21, C8/18	614[2]	528-701
	C6/22	2449	2449
1975	A7/5, A7/6, B7/6, C7/5, C7/6	42	28-68
	A7/19, A7/26, A7/27, B7/5, B7/26 C7/19, C7/20, C7/26, C8/2	169	107-228
	A7/20, A8/3, B7/27, D7/19, D8/2	324	273-372
	A8/2, B7/19, B7/20, B8/2, C7/27, D7/20	552	478-634
	B8/3, C8/3	1312[2]	800-1824

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island; A — 18th-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island; C — 2nd-4th Streets, Brighton, Coney Island.

[2]Arithmetic mean

TABLE A11. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO TOTAL COLIFORM DENSITIES

Year	Beach and Date[1]	Total Coliform Density/100 ml Mean	Range
1973	R7/14, R7/22, R7/28, R7/29, R8/11 R8/12, R8/18, R8/19	39.8	14-68
	C7/14, C7/22, C7/28, C7/29, C8/11 C8/12, C8/18, C8/19	983	256-5015
1974	R7/28, R8/18	28.0[2]	26-30
	R6/22, R7/20, R7/21	62.7	49-80
	C7/20, C7/21, C7/28	866	765-933
	C6/22, C8/18	2379[2]	1820-2938
1975	A7/6, C7/5, C7/6	109	92.9-141
	A7/5, A7/26, A7/27, B7/5, B7/6, B7/26	212	179-296
	A7/19, A8/2, A8/3, B7/20, B7/27 C7/19, C7/20, C7/26, C8/2, D8/2	576	391-765
	A7/20, B8/2, C7/27, D7/19, D8/3	1071	1007-1167
	B7/19, B8/3, C8/3, D7/20	2221	1332-3450

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island; A — 18th-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island; C — 2nd-4th Streets, Brighton, Coney Island.

[2]Arithmetic mean

TABLE A12. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO *Klebsiella* DENSITIES

Year	Beach and Date[1]	*Klebsiella* Density/100 ml Mean	Range
1973	R7/14, R7/22, R7/28, R7/29, R8/11 R8/12, R8/18, R8/19	13.7	1.2-15.3
	C7/14, C7/22, C7/28, C7/29, C8/11 C8/12, C8/18, C8/19	122	49-1006
1974	R6/22, R7/20, R7/21, R7/28, R8/18, R8/31	4.0	2-11
	C7/27, C8/31	16[3]	11-21
	C7/20, C7/21	45[3]	38-52
	C6/22, C8/18	336[3]	199-473
1975	A7/5, A7/6, B7/26, C7/5, C7/16 C7/19, C7/26	21.8	8.9-36
	A7/26, A7/27, B7/5, B7/6, B7/27 C7/27	57.6	49-67
	A7/19, A8/3, B7/20, C8/2	130	100-159
	A8/2, B7/19, C7/20, D8/2	203	182-214
	A7/20, B8/2, B8/3, C8/3, D7/19 D7/20, D8/3	378	235-1780[2]

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island; A — 18th-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island; C — 2nd-4th Streets, Brighton, Coney Island.

[2] All but one trial in range 235-389.

[3] Arithmetic mean

TABLE A13. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO *Enterobacter-Citrobacter* DENSITIES

Year	Beach and Date[1]	*Entero-Citro.* Density/100 ml Mean	Range
1973	R7/14, R7/22, R7/28, R7/29, R8/11 R8/12, R8/18, R8/19	11.1	1-24
	C7/14, C7/22, C7/28, C7/29, C8/11 C8/12, C8/18, C8/19	530	333-3612
1974	R7/28, R8/18	2.0	2
	R7/21, R8/31	7.5[2]	6-9
	R6/22, R7/20	20.0[2]	19-21
	C7/20, C7/21, C7/28	316	281-364
	C8/18, C8/31	485[2]	459-511
	C6/22	935	935
1975	A7/6, B7/5, B7/6, C7/5, C7/6	35.5	60-92
	A7/5, A7/26, A7/27, A8/3, B7/26 B7/27, C7/19, C8/2, D7/19	224	152-318
	A7/19, A7/20, A8/2, B7/20, C7/20, C7/26	376	338-407
	B8/2, C7/27, D8/2, D8/3	606	476-735
	B7/19, B8/3, C8/3, D7/20	1269	941-1979

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island; A — 18th-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island; C — 2nd-4th Streets, Brighton, Coney Island.

[2] Arithmetic mean

TABLE A14. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO *P. aeruginosa* DENSITIES

Year	Beach and Date[1]	*P. aeruginosa* Density/100 ml Mean	Range
1973	R7/14, R7/22, R7/28, R7/29, R8/11 R8/12, R8/18, R8/19	6.5	0.3-11
	C7/14, C7/22, C7/28, C7/29, C8/11 C8/12, C8/18, C8/19	30.4	8-45
1974	R7/20, R8/18, R8/31	2.0	0-4
	R7/21, R7/28	6.0	6
	C7/20, C8/18, C8/31	22.0	16-24
	C7/28	60.0	60
	C7/21	377	377
1975	A7/19, A7/26, A7/27, B7/6, B7/26, C7/19	8.0	5.4-13.5
	A7/6, B7/19, B7/27, C7/6, C7/26	19.5	16.2-24.6
	A8/2, C7/20, C8/2, D8/2	34.2	30.2-37.2
	A7/20, A8/3, C7/27, D8/3	60.7	50.1-77.7
	B7/20, B8/2, B8/3, C8/3	173	100-661[2]

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island; A — 18th-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island; C — 2nd-4th Streets, Brighton, Coney Island.
[2] All but one trial in range 100-126.

TABLE A15. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO *A. hydrophila* DENSITIES

Year	Beach and Date[1]	*A. hydrophila* Density/100 ml Mean	Range
1973	R7/14, R7/22, R7/28, R7/29, R8/11 R8/12, R8/18, R8/19	26.5	1-39
	C7/14, C7/22, C7/28, C7/29, C8/11 C8/12, C8/18, C8/19	25.3	1-244
1974	R7/20, R7/21, R7/28	1.7	1-3
	C7/20, C7/28, C8/31	5.0	5
	R8/18, C7/21	8.5[2]	7-10
	R6/22, R8/31, C6/22, C8/18	25.8	20-33
1975	B7/5, C7/5	2.4[2]	2.0-2.9
	A7/5, B7/6, B7/26, B7/27, C7/6, C7/27	40.9	18-75
	A7/6, A8/2, C7/19, C7/20, C7/26 C8/2, C8/3	140	104-163
	A7/26, A7/27, A8/3, B7/19 B8-2, B8/3	412	221-723
	A7/19, A7/20, B7/20	1182	899-1740

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island; A — 18-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island; C — 2nd-4th Streets, Brighton, Coney Island.
[2] Arithmetic mean

TABLE A16. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO *C. perfringens* DENSITIES

Year	Beach and Date[1]	*C. perfringens* Density/100 ml Mean	Range
1974	R7/21, R7/28, R8/31, C7/21	3.8	2-5
	R8/18, C7/28, C8/18	10.3	10-11
	R7/20, C7/20, C8/31	32.7	24-47
	R6/22	351.	351
1975	A7/5, B7/27, C7/27	9.3	7.1-11
	A7/26, B7/6, C7/5	18.2	16-21
	A7/6, A7/27, A8/3, B7/5, B7/26 C7/6, C7/26, C8/3	28.7	25-33
	B7/19, B7/20, B8/3	68.6	48-91

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island; A — 18th-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island; C — 2nd-4th Streets, Brighton, Coney Island.

TABLE A17. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO *Staphylococcus* DENSITIES

Year	Beach and Date[1]	*Staphylococcus* Density/100 ml Mean	Range
1974	R8/31	32	32
	R8/18, C6/22, C8/31	112	98-137
	R6/22, R7/28, C7/28	189	177-210
	R7/20, C7/20, C8/18	344	303-398
	R7/21, C7/21	742[2]	558-926
1975	A7/5, A7/6, C7/5	11.7	5.5-32
	A7/19, A7/27, B7/5, B7/27, C7/6, C7/19	76.7	46-123
	B7/6, B7/19, B7/26, C7/20, C7/27	197	155-245
	A7/20, A7/26, A8/3, C8/2, C8/3	655	537-776
	A8/2, B7/20, B8/2, B8/3, C7/26	1572	955-4070

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island; A — 18th-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island; C — 2nd-4th Streets, Brighton, Coney Island.

[2] Arithmetic mean

TABLE A18. MEAN AND RANGE OF NEW YORK CITY TRIALS CLUSTERED ACCORDING TO *V. parahaemolyticus* DENSITIES

Year	Beach and Date[1]	*V. parahaemolyticus* Density/100 ml	
		Mean	Range
1974	R7/21, C7/21	9.5[2]	5-14
	R7/28, R8/18, C7/28, C8/18	36.6	28-61
	R8/31, C8/31	309[2]	249-368
1975	A7/5, A7/6, A7/27, B7/5, B7/6 B7/27, C7/5, C7/6, C7/27	3.8	1.5-10.6
	A7/19, A7/26, B7/19, B7/26, C7/19 C7/26, C8/2, C8/3	35.5	23-68
	A8/2, B7/20, B8/2, C7/20	121	82-189
	A7/20, A8/3, B8/3	444	431-463

[1] R — Rockaway; C — Coney Island; D — 34th-38th Streets, Coney Island;
A — 18th-24th Streets, Coney Island; B — 8th-10th Streets, Coney Island;
C — 2nd-4th Streets, Brighton, Coney Island.

[2] Arithmetic mean

TABLE A19. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY ENTEROCOCCUS DENSITIES

Year	Enterococcus Density/100 ml	N		Rate/GI Symptoms			Rate/HCGI Symptoms		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
				Per 1000 Persons			Per 1000 Persons		
1973	21.8	484	197	81	46	35	30	15	15
	91.2	474	167	72	24	48*	46	18	28
1974	3.6	1441	711	27	23	4	7.6	4.2	3.4
	7.0	951	1009	38	34	4	10.5	6.9	3.6
	13.5	625	419	42	17	25*	16.0	2.4	13.6
	31.5	831	440	43	23	20	18.1	—	18.1*
1975	5.7	2232	935	63	55	8	18.8	19.3	−0.5
	20.3	1896	678	59	37	22*	14.8	7.4	7.4
	154	579	191	60	31	29	34.5	—	34.5*

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*p<.05; **p<.01.

Note: Yearly totals for values of N on Tables A19-A28 may not agree with those in Table 3 because data for one or more indicators were not availble for all trial dates.

TABLE A20. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY *E. coli* DENSITIES

Year	*E. coli* Density/100 ml	N		Rate/GI Symptoms			Rate/HCGI Symptoms		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
				Per 1000 Persons			Per 1000 Persons		
1973	24.8	484	197	81	46	35	30	15	15
	174	474	167	72	24	48*	46	18	28
1974	2.5	2514	1641	25	34	−9	8.0	3.7	4.3
	13.8	1304	1045	38	29	9	14.1	5.7	8.4*
1975	30.5	600	425	65	33	32*	23.3	2.4	20.9*
	46.8	1945	1099	55	51	4	13.4	17.8	−4.4
	142	775	194	76	41	35	24.5	10.3	14.2
	278	1049	330	55	24	31*	21.0	3.0	18.0*
	514	937	271	68	55	13	24.5	7.4	17.1

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*$p<.05$; **$p<.01$.

TABLE A21. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY FECAL COLIFORM DENSITIES

Year	Fecal Coliform Density/100 ml	N		Rate/GI Symptoms Per 1000 Persons			Rate/HCGI Symptoms Per 1000 Persons		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
1973	21.6	484	197	81	46	35	30	15	15
	165	474	167	72	24	48*	46	18	28
1974	18.0	958	472	35	34	1	10.4	6.4	4.0
	39.0	1133	1246	48	43	5	10.7	6.4	4.3
	252	625	419	42	17	25*	16.0	2.4	13.6
	614	831	440	43	23	20	18.1	—	18.1*
	2449	236	184	72	49	23	21.2	5.4	15.8
1975	41.6	1131	472	69	57	12	16	23	−7.0
	169	1457	680	55	44	11	13	13	0
	324	724	223	54	27	27	22	4.5	17.5
	552	1123	333	62	48	14	24	6.0	18.0
	1312	292	96	72	31	41	34.5	—	34.5

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*p<.05; **p<.01.

TABLE A22. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY TOTAL COLIFORM DENSITIES

Year	Total Coliform Density/100 ml	N		Rate/GI Symptoms Per 1000 Persons			Rate/HCGI Symptoms Per 1000 Persons		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
1973	39.8	487	197	81	46	35	30	15	15
	983	474	167	72	24	48*	46	18	28
1974	28.0	958	472	35	34	1	10.4	6.4	4.0
	62.7	1133	1246	48	43	5	10.6	6.4	4.2
	866	1086	719	44	22	22*	16.6	1.4	15.2**
	2380	606	324	51	31	20	19.8	3.1	16.7
1975	109	717	318	56	54	2	12.6	12.6	0.0
	212	1074	597	58	50	8	13.0	20.1	−7.1
	576	1618	478	62	34	28**	22.2	8.4	13.8
	1071	694	229	69	57	12	20.2	8.7	11.5
	2221	604	182	63	33	30	28.1	5.5	22.6

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*$p<.05$; **$p<.01$.

TABLE A23. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY *Klebsiella* DENSITIES

Year	*Klebsiella* Density/100 ml	N		Rate/GI Symptoms			Rate/HCGI Symptoms		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
				Per 1000 Persons			Per 1000 Persons		
1973	13.7	484	197	81	46	35	30	15	15
	122	474	167	72	24	48	46	18	28
1974	3.7	2767	2156	39	35	4	11.9	11.6	0.3
	18.0	463	289	17	21	−4	2.2	3.4	−1.2
	45.0	825	541	53	26	27	20.5	—	20.5
	336	606	324	51	31	20	19.8	3.1	16.7
1975	22.0	1475	607	62	51	11	11.5	21.4	−9.9
	58.0	1182	668	55	49	6	16.9	12.0	4.9
	130	566	148	64	20	44	24.7	—	24.7
	203	633	136	65	44	21	30.0	22.1	7.9
	378	841	245	65	37	28	23.8	4.1	19.7

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*p<.05; **p<.01.

TABLE A24. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY *Enterobacter-Citrobacter* DENSITIES

Year	*Entero.-Citro.* Density/100 ml	N		Rate/GI Symptoms			Rate/HCGI Symptoms		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
				Per 1000 Persons			Per 1000 Persons		
1973	11.1	484	197	81	46	35	30	15	15
	530	474	167	72	24	48**	46	18	28*
1974	2.0	958	472	35	34	1	10.4	6.4	4.0
	7.5	970	710	27	17	10	8.2	2.8	5.4
	20.0	596	775	54	53	1	8.4	7.7	0.7
	316	1086	719	44	22	22	16.6	1.4	15.2
	485	572	251	31	20	11	12.2	—	12.2
	935	236	184	72	45	27	21.2	5.4	15.8
1975	35.5	1136	560	59	48	11	14.1	16.1	−2.0
	224	1652	616	56	46	10	12.1	14.6	−2.5
	376	725	276	59	40	19	29.0	14.5	14.5
	606	590	170	80	59	21	27.1	5.9	21.2
	1269	604	182	63	33	30	28.1	5.5	22.6

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*$p<.05$; **$p<.01$.

TABLE A25. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY *P. aeruginosa* DENSITIES

Year	*P. aeruginosa* Density/100 ml	N		Rate/GI Symptoms Per 1000 Persons			Rate/HCGI Symptoms Per 1000 Persons		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
1973	6.5	484	197	81	46	35	30	15	15
	30.4	474	167	72	24	48**	46	18	28
1974	2.0	1277	879	30	28	2	9.4	8.0	1.4
	6.0	873	730	37	29	8	10.4	4.1	6.3
	22.0	936	492	43	20	23	17.1	—	17.1
	60.0	261	178	15	11	4	3.8	5.6	−1.8
	377.0	461	300	48	30	18	17.4	—	17.4
1975	8.0	1097	480	58	60	−2	13.7	20.8	−7.1
	19.5	1111	448	43	38	5	18.9	11.1	7.8
	34.2	543	116	76	35	41	23.9	17.5	6.7
	60.7	389	182	72	55	17	20.6	5.5	15.5
	173	736	192	68	37	31	32.6	—	32.6

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*p<.05, **p<.01.

TABLE A26. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY *A. hydrophila* DENSITIES

Year	*A. hydrophila* Density/100 ml	N		Rate/GI Symptoms Per 1000 Persons			Rate/HCGI Symptoms Per 1000 Persons		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
1973	26.5	484	197	81	46	35	30	15	15
	25.3	474	167	72	24	48**	46	18	28*
1974	1.7	1085	1157	39	34	5	11.1	6.9	4.2
	5.0	827	530	36	21	15	12.1	1.9	10.2
	8.5	1083	513	42	31	11	12.9	3.9	9.0
	25.8	1423	911	40	36	4	10.5	2.2	8.3
1975	2.4	471	251	66	40	26	8.5	8.0	0.5
	40.9	1280	580	63	53	10	14.8	20.7	−5.9
	140	1076	365	69	38	31	21.4	13.7	7.7
	412	1210	403	53	52	1	21.5	7.4	14.1
	1182	322	109	71	28	43	34.2	—	34.2

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*$p < .05$; **$p < .01$.

TABLE A27. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY *C. perfringens* DENSITIES

Year	*C. perfringens* Density/100 ml	N		Rate/GI Symptoms			Rate/HCGI Symptoms		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
				Per 1000 Persons			Per 1000 Persons		
1974	3.8	1767	1269	33	24	9	10.2	2.4	7.8
	10.3	1253	531	34	19	15	12.8	6.0	6.8
	32.7	778	779	46	36	10	15.4	6.4	9.0
	351.	384	348	57	66	−9	2.6	5.7	−3.1
1975	9.3	617	267	75	45	30	19.4	11.2	8.2
	18.2	699	312	64	67	−3	14.3	25.6	−11.3
	28.7	1178	713	48	41	7	17.8	11.2	6.6
	68.6	607	172	61	41	20	31.3	5.8	25.5

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*p<.05; **p<.01.

TABLE A28. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY *Staphylococcus* DENSITIES

Year	*Staphylococcus* Density/100 ml	N		Rate/GI Symptoms			Rate/HCGI Symptoms		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
				Per 1000 Persons			Per 1000 Persons		
1974	32.0	433	239	9.2	—	9.2	2.3	—	2.3
	112	1060	508	43	39	4	12.3	5.9	6.4
	189	1081	785	33	43	−10	4.6	3.8	0.8
	344	946	808	49	30	19	20.1	6.2	13.9
	719	998	949	44	24	20	15.0	2.1	12.9
1975	11.7	631	251	76	52	24	17.4	19.9	−2.5
	76.9	1175	544	57	39	18	12.8	3.7	9.1
	197	947	399	66	65	1	23.2	30.1	−6.9
	655	660	224	58	31	27	16.7	4.5	12.2
	1572	946	290	58	41	17	25.4	6.9	18.5

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*p<.05; **p<.01.

TABLE A29. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GI (HCGI) SYMPTOM RATES FOR NEW YORK CITY TRIALS CLUSTERED BY *V. parahaemolyticus* DENSITIES

Year	*V. parahaemolyticus* Density/100 ml	N		Rate/GI Symptoms Per 1000 Persons			Rate/HCGI Symptoms Per 1000 Persons		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
1974	8.4	998	771	44	27	17	15	2.6	12.4
	36.2	1589	800	33	24	9	11.3	5.0	6.3
	303	635	350	13	11	2	1.6	—	1.6
1975	3.8	1907	939	64	50	14	16.3	13.8	2.5
	35.5	1369	468	57	41	16	16.1	15.0	1.1
	121	674	169	65	47	18	31.2	12.5	18.7
	444	390	132	64	38	26	23.1	—	23.1

[1] Swimmer.
[2] Nonswimmer.
[3] Swimming-Associated (swimmer-nonswimmer).
*p<.05; **p<.01.

TABLE A30. MEAN AND RANGE OF INDICATOR DENSITIES FOR ALL TRIALS CONDUCTED DURING A GIVEN YEAR (SUMMER), NEW YORK CITY BEACHES

Year	Beach	Indicator Density Per 100 ml: Enterococci Mean	Enterococci Range	*Escherichia coli* Mean	*Escherichia coli* Range	Fecal Coliforms Mean	Fecal Coliforms Range	Total Coliforms Mean	Total Coliforms Range
1973	Rockaways	21.8	5-30	24.8	12-34	21.5	10-31	39.8	22-68
	Coney Island	91.2	23-186	174	40-709	165	49-431	983	256-5015
1974	Rockaways	3.5	2-7	2.4	1-9	28.4	17-50	43.2	26-80
	Coney Island	16.4	7-33	15.3	4-35	565	231-2449	1213	765-2938
1975[1]	Coney Island(C)	17.9	6-199	52.4	22-506	134	37-585	426	93-1920
	Coney Island(B)	27.7	6-298	98.6	60-659	359	68-1824	633	179-3450
	Coney Island(A)	6.7	2-88	61.3	23-318	130	28-478	844	141-1167
	Coney Island(D)	14.2	8-26	157	137-292	405	326-565	1050	765-1332

Year	Beach	Indicator Density Per 100 ml: *Klebsiella sp.* Mean	*Klebsiella sp.* Range	*Enter.-Citro.* Mean	*Enter.-Citro.* Range	*P. aeruginosa* Mean	*P. aeruginosa* Range	*A. hydrophila* Mean	*A. hydrophila* Range
1973	Rockaways	13.7	1-15	11.1	1-24	6.5	1-11	26.5	2-39
	Coney Island	122	49-260	530	123-3612	30.4	8-45	25.3	1-244
1974	Rockaways	3.5	2-5	6.6	2-21	3.1	0-6	4.9	1-33
	Coney Island	59.2	11-473	434	93-281	45.8	16-377	9.6	5-27
1975[1]	Coney Island(C)	56.4	9-288	288	63-140	26.6	9-126	63.2	2-163
	Coney Island(B)	126	30-1780	348	89-197	47.7	10-661	124	3-899
	Coney Island(A)	75	16-280	204	60-377	16.7	5.4-66	216	18-1740
	Coney Island(D)	279	209-389	545	94-318	51.9	35-78	ND	ND

[1] Coney Island Beaches: (C) 2nd-4th Streets, Brighton; (B) 8th-10th Streets; (A) 18th-24th Streets; (D) 34th-38th Streets.

TABLE A30. (Continued)

Year	Beach	Indicator Density Per 100 ml *C. perfringens* Mean	*C. perfringens* Range	*V. parahaemolyticus* Mean	*V. parahaemolyticus* Range	*Staphylococci* Mean	*Staphylococci* Range
1973	Rockaways	ND	ND	ND	ND	ND	ND
	Coney Island	ND	ND	ND	ND	ND	ND
1974	Rockaways	12.5	2-351	32.8	5-249	178	32-558
	Coney Island	18.3	50-66	54.5	14-368	243	98-926
1975[1]	Coney Island(C)	17.5	7.1-32	16.1	3.0-100	209	32-955
	Coney Island(B)	21.6	10-91	34.2	1.5-463	378	96-4370
	Coney Island(A)	22.6	12-33	41.5	3.3-438	128	5.5-219
	Coney Island(D)	ND	ND	ND	ND	ND	ND

[1] Coney Island Beaches: (C) 2nd-4th Streets, Brighton; (B) 8th-10th Streets; (A) 18th-24th Streets; (D) 34th-38th Streets.

TABLE A31. GASTROINTESTINAL (GI) AND HIGHLY CREDIBLE GASTROINTESTINAL (HCGI) SYMPTOM RATES BY BEACH AND YEAR FOR NEW YORK CITY TRIALS

Year	Beach	N		Rate of GI Symptoms Per 1000 Persons			Rate of HCGI Symptoms Per 1000 Persons		
		Swim[1]	Nonswim[2]	Swim	Nonswim	▲[3]	Swim	Nonswim	▲
1973	Rockaway	484	197	81	46	35	30	15	15
	Coney Is.	474	167	72	24	48*	46	18	28
1974	Rockaway	2767	2156	39	35	4	12	12	0
	Coney Is.	1961	1185	42	26	16*	16	9.3	6.7
1975	Coney Is. (C)	1534	590	70	54	16	21.2	12.6	8.6
	Coney Is. (B)	1744	623	57	42	15	21.8	22.5	−0.7
	Coney Is. (A)	1131	475	50	44	6	13.7	8.5	4.2
	Coney Is. (D)	298	96	60	31	29	23.5	10.4	13.1

[1] Swimmer
[2] Nonswimmer
[3] Swimming-Associated (swimmer-nonswimmer)

*p< .05 **p< .01

TABLE A47. GASTROINTESTINAL SYMPTOM RATES AND CORRESPONDING MEAN INDICATOR DENSITIES FOR 1978 TRIALS AT LAKE PONTCHARTRAIN

Indicator	Density/100 ml		N		Rate for 1000 Persons for Gastrointestinal Symptoms			Rate for 1000 Persons for Highly Credible GI Symptoms		
	Mean	Range	Swim	Nonswim	Swim	Nonswim	▲	Swim	Nonswim	▲
Enterococcus	11.1	3-30	1230	415	75	34	41**	36.6	14.5	22.1*
	14.4 [1]	3-325	248	303	82	63	18	44.3	23.1	21.3
	142	67-303	801	322	112	50	62**	42.4	15.5	26.9*
E. coli	9.0 [1]	1-23	248	303	81	63	18	44.3	23.1	21.3
	32.6	17-87	1123	383	78	44	33*	38.3	20.9	17.4
	93.7	53-177	918	355	103	36	67**	39.2	8.5	30.7*

[1] Fontainebleau Beach.
*p<0.05; **p<0.01

TABLE A48. SYMPTOM RATES FOR REVERE AND NAHANT BEACHES DURING 1978 BOSTON HARBOR STUDY

Symptom Group	Rate Per 1000 Persons At Revere Beach[1]			Rate Per 1000 Persons At Nahant Beach[2]		
	Swim	Nonswim	▲	Swim	Nonswim	▲
Gastrointestinal	89.0	70.0	19.0	69.6	63.7	5.9
Respiratory	82.7	72.3	10.4	98.2	102.8	−4.6
Other	83.8	68.5	14.3	82.3	102.8	−20.5
Ear, Eyes, Nose	95.8	99.0	−3.2	87.7	109.2	−21.4
Highly Credible GI	27.0	12.0	15.0	33.0	28.0	5.0
Severe GI	34.8	29.8	5.0	26.5	28.2	−1.7

[1] N—919 swimmers; 905 nonswimmers.
[2] N—1150 swimmers; 1099 nonswimmers.

TABLE A49. GASTROINTESTINAL SYMPTOM RATES AND CORRESPONDING INDICATOR DENSITIES FOR REVERE AND NAHANT BEACHES FOR 1978 BOSTON HARBOR STUDY

Beach	Indicator Density/100 ml				Rate for GI[1] Symptoms Per 1000 Persons			Rate for HCGI[2] Symptoms Per 1000 Persons		
	Enterococci		*E.coli*							
	Mean	Range	Mean	Range	Swim[3]	Nonswim[4]	▲	Swim	Nonswim	▲
Revere[5]	6.3	2-12	18.0	5-31	89	70	19	27	10	15
Nahant[6]	7.3	6-9	11.5	4-22	70	64	6	33	28	5

[1] Gastrointestinal; [2] highly credible gastrointestinal; [3] swimmers; [4] nonswimmers; [5] data from four trials (days); N for swimmers 919, for nonswimmers 905; [6] data from four trials (days); N for swimmers 1150, for nonswimmers 1099.

TABLE A50. GASTROINTESTINAL SYMPTOM RATES AND CORRESPONDING INDICATOR DENSITIES FOR CLUSTERED TRIALS DURING 1978 BOSTON HARBOR STUDY

Indicator	Density/100 ml		Beach	N		Gastrointestinal Symptoms Per 1000 Persons			Highly Credible Symptoms Per 1000 Persons		
	Mean	Range		Swim	Nonswim	Swim	Nonswim	▲	Swim	Nonswim	▲
Enteroccocci	4.3[1]	2-6	Revere	697	529	83	66	17	23	11	12
	7.3[2]	6-9	Nahant	1130	1099	71	67	4	33	28	5
	12.0[3]	12	Revere	222	376	108	74	34*	41	13	28*
E. coli	5.5[4]	4-7	Nahant	541	874	72	63	9	39	29	10
	7.0[5]	5-9	Revere	477	410	86	68	18	23	9.8	13
	17.5[6]	13-22	Nahant	589	225	70	67	3	27	27	0
	29.5[7]	28-31	Revere	442	495	93	71	22	32	14	17

Trials clustered—[1] 1, 3, 4; [2] 1, 2; [3] 2; [4] 1, 2; [5] 1, 3; [6] 3, 4; [7] 2, 4.
*$p<0.05$.

TABLE A51. 95% CONFIDENCE LIMITS FOR SWIMMING-ASSOCIATED GASTROINTESTINAL SYMPTOM RATES PREDICTED FROM THE OBSERVED MEAN ENTEROCOCCUS DENSITIES (TRIALS CLUSTERED BY INDICATOR DENSITIES)

Enterococcus Density Per 100 ml	Total GI Symptoms			HCGI[1] Symptoms		
	Predict.[2] Rate	95% Conf. Lim. Lower	Upper	Predict. Rate	95% Conf. Lim. Lower	Upper
3.6	8.4	−0.8	17.6	6.9	1.0	12.8
4.3	10.2	1.6	18.9	7.8	2.3	13.4
5.7	13.2	5.3	21.0	9.3	4.3	14.4
7.0	15.4	8.0	22.7	10.4	5.7	15.1
7.3	15.8	8.6	23.0	10.7	6.0	15.3
11.1	20.2	13.9	26.4	12.9	8.9	16.9
12.0	21.0	14.9	27.1	13.3	9.0	17.2
13.5	22.3	16.3	28.2	13.9	10.1	17.7
14.4	22.9	17.1	28.8	14.3	10.5	18.0
20.3	26.5	21.1	32.0	16.1	12.6	19.6
21.8	27.3	21.8	32.7	16.5	13.0	20.0
31.5	31.2	25.7	36.7	18.4	14.9	22.0
44.0	34.7	28.8	40.5	20.2	16.5	24.0
91.2	42.3	35.0	49.7	24.1	19.4	28.8
142.0	47.0	38.4	55.5	26.4	21.0	31.9
154.0	47.8	39.0	56.3	26.9	21.2	32.5
224.0	51.8	41.8	61.7	28.9	22.5	35.3
495.0	60.1	47.5	72.7	33.1	25.0	41.2

[1] Highly credible gastrointestinal.
[2] Rates predicted from Y on X regression lines.

TABLE A52. 95% CONFIDENCE LIMITS FOR MEAN ENTEROCOCCUS DENSITIES PREDICTED FROM THE OBSERVED SWIMMING-ASSOCIATED GI SYMPTOM RATES

Total GI Per 1000 Persons	Enterococcus Density/100 ml			HCGI[1] Per 1000 Persons	Enterococcus Density/100 ml		
	Predict.[2]	95% Conf. Lim. Lower	Upper		Predict.	95% Conf. Lim. Lower	Upper
4	5.1	2.5	10.4	−0.5	3.8	1.4	10.3
8	6.6	3.4	12.5	3.4	5.7	2.5	13.3
17	11.7	7.1	19.5	3.6	5.9	2.5	13.5
18	12.5	7.6	20.5	5.0	6.8	3.1	14.8
20	14.2	8.9	22.9	7.4	8.8	4.4	17.5
22	16.2	10.3	25.5	12.0	14.4	8.2	25.1
25	19.7	12.7	30.4	13.6	17.0	10.1	28.8
29	29.0	16.6	39.0	15.2	20.2	12.2	33.5
34	35.2	22.6	54.8	18.1	27.6	16.8	45.2
35	37.5	23.9	58.9	20.9	37.2	22.0	62.9
41	55.4	33.1	92.6	21.2	38.4	22.6	65.3
48	87.1	46.8	162.0	22.1	42.3	24.4	73.2
54	128.0	61.9	266.0	23.1	47.1	26.6	83.4
58	166.0	74.0	373.0	26.9	70.6	35.6	140.0
62	215.0	88.4	524.0	27.2	72.9	36.3	146.0
				28.0	79.5	38.5	164.0
				28.4	82.9	39.6	174.0
				34.5	159.0	59.5	426.0

[1] Highly credible GI symptom.
[2] Predicted from X on Y regression lines.

* U.S. GOVERNMENT PRINTING OFFICE: 1983- 659-095/0730